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# TWELVE YEARS' EXPERIENCE IN ROENTGENOTHERAPY FOR CHRONIC ARTHRITIS*

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ROENTGENOTHERAPY as a local treatment to the joints in chronic arthritis was begun by us over twelve years ago. We were prompted to institute this form of treatment after reading the early optimistic reports of Kahlmeter[1] in Sweden and of Scott[2] in London. At first the treatments were given with great caution. After a number of unexpected successes and an equal number of failures, a general plan of treatment for certain articular lesions in chronic arthritis was developed. During this time we learned to expect certain effects in the joints, and by trial and error we found some of the indications and contraindications to the treatment. By the end of August, 1945, x-ray therapy had been given to 370 selected patients, of whom 252 had rheumatoid arthritis and 118 osteoarthritis. Five hundred and sixteen separate joints were treated, exclusive of spines. In the majority of cases one joint was treated. Although a large number of patients were treated for various types of nonarticular rheumatic disease, such as fibrositis and bursitis, these have not been included in this study.

The articular lesions of chronic arthritis have been treated with x-ray almost from the time of Roentgen's discovery in 1896. In 1897 Sokolow[3] published the first report on this treatment, claiming that it gave relief in arthritis deformans. The first American report was made in 1906 by Anders, Daland and Pfahler.[4] Since the beginning of the century numerous studies have appeared and, almost without exception, have claimed improvement in chronic arthritis following roentgenotherapy. Staunig[5] reported in 1925 that he had treated 400 patients. In 1933 Langer,[6] stating that therapy had been beneficial in 200 cases, advocated x-ray treatment to the spinal ganglions, since he believed that one of the main actions was on the sympathetic nervous system. By 1935 2000 arthritic patients had been treated by von Pannevitz,[7] who reported improvement in more than 95 per cent. Between 1925 and 1937 Kahlmeter[8] treated over 5000 patients; although excellent results were claimed in the entire group, he stated that the best results were seen in the early stages of arthritis. Scott,[9] who was one of the pioneers in roentgenotherapy for arthritis, in 1939 advocated wide-field x-ray therapy, which he reported to be particularly valuable in rheumatoid arthritis of the spine.

The first carefully controlled study on the effect of roentgenotherapy in chronic arthritis was made in 1941 by Smyth, Freyberg and Peck[10] on 100 unselected patients with various forms of rheumatic disease. They concluded that significant improvement that could be attributed to the therapy occurred in only 14 per cent of patients. Definite relief, however, followed roentgenotherapy in 72 per cent of patients with rheumatoid arthritis of the spine. They believed that much of the improvement reported by earlier writers was largely subjective. In the earlier papers little in the way of statistics or objective data was given. The findings were difficult to evaluate because of confusion of local with systemic changes and of subjective with objective improvement. The suggestive effect of this form of therapy on certain patients is often profound. For this reason subjective impressions must always be viewed with suspicion. The x-rays undoubtedly exert some systemic effect, but such systemic changes are inconstant and usually immeasurable. These varied reports on the value of roentgenotherapy led us to analyze our objective and measurable findings in the treatment of a selected group of patients suffering from chronic arthritis.

## METHOD OF TREATMENT

Our technic of treatment was similar to that reported from both European and American clinics. The method varied in different joints, depending on the tissues to be penetrated and on the anatomic area to be covered. As a rule 200 r was given twice a week over two to four target areas for six treat-

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ments; 1200 r in six treatments during a period of three weeks comprised a course of treatment, and no further treatments were given to the patient until at least two months had elapsed. In rheumatoid arthritis of the spine, wide-field therapy in doses of 100 r was applied over two spinal areas, an upper and a lower, every other day for twelve treatments; no further therapy was given for two months. The dosage depended on the stage of the disease and the general condition of the patient.

At first an arthritic inflammation that showed little response to rest and physiotherapy was the only indication for roentgenotherapy. Before long, however, it was noticed that certain lesions responded fairly readily to x-ray treatment whereas others did not, and roentgenotherapy was therefore prescribed more carefully. The selected group of 370 patients was about one tenth of the total treated on the wards during the twelve-year period.

Since articular changes occur frequently with exacerbations and remissions in the disease, it is essential that patients be observed during a period in which their condition remains relatively unchanged. In such cases x-ray therapy was used as an adjunct to the general medical and orthopedic measures. The patients were compared with an equal number on the wards at the same time who had similar articular lesions whose treatment differed only in the absence of roentgenotherapy. Only changes in the joints that were more rapid and definite in the patients treated with x-ray were recorded as improved; when the changes were no different in the treated and untreated groups the effect of the roentgenotherapy was recorded as negative.

Objective changes were evaluated by observation, by measurement of the circumference of the joint and by recording the range of active, painless motion. If decrease in swelling and tenderness was little, with a decrease in articular circumference of less than 1.3 cm. and with an increased range of active, painless motion no greater than 10°, the improvement was recorded as slight. If the decrease shown by articular circumference was from 1.3 to 2.5 cm. and there was an increased range of painless, active motion of from 10 to 30°, improvement was recorded as moderate; the effect beyond this was tabulated as marked. Improvement in the spine, although definite, was more difficult to evaluate. Increase in chest expansion and more rapid correction of the forward bowing of the spine were the most important changes observed. Subjective improvement was recorded by questioning the patient about the amount of pain, how freely motions could be performed and how much stiffness was present.

## Results

The results of roentgenotherapy in this selected group of patients are shown in Table 1. In most cases the subjective impressions of change were almost parallel to the objective, and although examination failed to verify the improvement claimed by some patients, the number of such cases was relatively small. Of the 290 cases in which objective changes were observed (78.4 per cent of the series) improvement was slight in 89 (24.1 per cent), moderate in 138 (37.2 per cent) and marked in 63 (17.1 per cent). In only 25 of the patients listed as markedly improved, was there complete subsidence of all inflammation and of all symptoms, a rare effect following roentgenotherapy. In 80 patients, or 21.6 per cent, no improvement could be found; this large percentage of failures was probably due chiefly to a faulty selection of articular lesions. Indiscriminate use of roentgenotherapy in all forms of rheumatic disease gave discouraging results, as Smyth, Freyberg and Peck[10] have shown.

Table 1. *Effect of Roentgenotherapy in 370 Selected Patients with Chronic Arthritis.*

| Basis | No Change | | Slight Improvement | | Moderate Improvement | | Marked Improvement | |
|---|---|---|---|---|---|---|---|---|
| | No. of cases | Per-centage | No. of cases | Per-centage | No. of cases | Per-centage | No. of cases | Per-centage |
| Subjective impression | 69 | 18.9 | 94 | 25.4 | 163 | 44.0 | 44 | 11.8 |
| Objective impression | 80 | 21.6 | 89 | 24.1 | 138 | 37.2 | 63 | 17.1 |

There were considerable variations in the effects of therapy in the different types of chronic arthritis (Table 2). The best results were obtained in rheumatoid arthritis of the spine, in which — as pointed out by Scott,[9] Hare[11] and Smyth, Freyberg and Lampe[12] — there is a more rapid subsidence of muscular spasm and pain, an increase in chest expansion and in spinal movement and a decrease or sometimes a complete disappearance of nerve-root pain. Improvement occurred in 84.6 per cent of the 98 patients with rheumatoid arthritis of the spine. Improvement was slight in 34 (34.7 per cent), moderate in 37 (37.7 per cent) and marked in 12 (12.2 per cent). Twelve patients were completely relieved of symptoms; 15 who were not improved showed marked ankyloses and deformities. In most cases, plaster casts were applied and active exercises were given during or soon after roentgenotherapy, usually with continued improvement. There were only 2 relapses. In rheumatoid arthritis without spondylitis, improvement was seen in 79.5 per cent of 154 patients. Improvement was slight in 26 (16.8 per

cent), moderate in 61 (39.9 per cent) and marked in 35 (22.7 per cent). Only 7 patients had complete subsidence of symptoms. The poorest results were obtained in osteoarthritis, but with a selection of the type of joint suitable for roentgenotherapy a fair response was obtained. The improvement occurring in 72.0 per cent of the 118 patients was slight in 29 (24.5 per cent), moderate in 40 (34.0 per cent) and marked in 16 (13.5 per cent). In 6 cases there was complete disappearance of symptoms. There were failures in 33 patients, most of whom had deformities of varying degree, as well as extensive spur formation. Mechanical disalignment, which seems to be more significant in osteoarthritis than in rheumatoid arthritis, continues to produce inflammatory changes in the joint, and little can be expected from local therapy until such disalignment has been at least partially corrected.

TABLE 2. *Effects of Roentgenotherapy in Different Types of Arthritis.*

| DIAGNOSIS | TOTAL NO. OF CASES | NO CHANGE | | SLIGHT IMPROVEMENT | | MODERATE IMPROVEMENT | | MARKED IMPROVEMENT | |
|---|---|---|---|---|---|---|---|---|---|
| | | NO. OF CASES | PERCENTAGE | NO. OF CASES | PERCENTAGE | NO. OF CASES | PERCENTAGE | NO. OF CASES | PERCENTAGE |
| Rheumatoid arthritis without spondylitis | 154 | 32 | 20.8 | 26 | 16.8 | 61 | 39.9 | 35 | 22.7 |
| Rheumatoid arthritis of spine | 98 | 15 | 15.4 | 34 | 34.7 | 37 | 37.7 | 12 | 12.2 |
| Osteoarthritis | 118 | 33 | 28.0 | 29 | 24.5 | 40 | 34.0 | 16 | 13.5 |

The selection of articular lesions for roentgenotherapy comprised about 1 in 12 of the patients receiving treatment on the wards. X-ray treatment was regarded not as a cure for arthritis, but as a helpful local measure in certain cases. The chief indication for roentgenotherapy in rheumatoid arthritis was chronic swelling in a joint that failed to subside after several weeks' rest and physiotherapy. Most of these joints respond to roentgenotherapy if there is no serious deformity or internal derangement. Persistent stiffness and pain were the indications in osteoarthritis, in which decrease in swelling is often difficult to measure and the effect of x-ray therapy seems to be chiefly one of analgesia with an increased range of motion at the joint. In rheumatoid arthritis of the spine, a decreased range of chest expansion and severe muscular spasm, with marked forward bowing of the spine, responded fairly readily to x-ray treatment and permitted earlier and more rapid application of corrective apparatus. Roentgenotherapy after manipulative procedures, which was given only during the last year of the study, seemed to lessen muscular spasm and to prevent the reformation of adhesions, but its action was observed for too short a time to justify a definite statement. None of the cases in which such therapy was used after manipulation were included in this study.

To determine the effect of suggestion on the changes observed, the treatments were given to one knee only in 10 patients with practically equal involvement of both knees. The x-ray apparatus and the draping were so arranged that the patients thought that both knees were being treated. The results of these treatments are given in Table 3. Only 1 patient reported that both knees felt better after the treatments, and no objective changes were found in any of the untreated knees. Definite objective improvement was observed in the treated knees in 7 cases. In 3 patients with severe deformity there were no objective changes, and only questionable subjective changes in 1. The psychic effect of roentgenotherapy in such patients therefore does not appear to have been an important factor in the improvement.

Kahlmeter[8] stated that better results were obtained with roentgenotherapy in the early than in other stages of chronic arthritis. A graph was made of all patients suffering from rheumatoid arthritis (Fig. 1). Although there is a slight decline of the curve with longer duration of symptoms, the variation from year to year is not great. It is probable that the decrease in improvement was due more to the greater number of severe deformities than to the duration of symptoms per se. With a local treatment like roentgenotherapy the condition of the joint at the time of treatment appeared to be the important factor. A comparison of the different types of chronic arthritis showed no significant variation from the graph for cases of rheumatoid arthritis, except that in osteoarthritis the response showed no relation to the duration of symptoms.

TABLE 3. *The Subjective Effect of Roentgenotherapy.*

| SEX | AGE | TREATED KNEE | | UNTREATED KNEE | |
|---|---|---|---|---|---|
| | | OBJECTIVE IMPROVEMENT | SUBJECTIVE IMPROVEMENT | OBJECTIVE IMPROVEMENT | SUBJECTIVE IMPROVEMENT |
| | yr. | | | | |
| F | 18 | ++ | ++ | 0 | 0 |
| M | 22 | ++ | ++ | 0 | 0 |
| M | 26 | ++ | + | 0 | 0 |
| F | 42 | 0 | 0 | 0 | 0 |
| F | 43 | 0 | + | 0 | 0 |
| F | 43 | 0 | 0 | 0 | 0 |
| F | 48 | +++ | ++ | 0 | 0 |
| F | 49 | ++ | +++ | 0 | ++ |
| F | 53 | + | ++ | 0 | 0 |
| F | 56 | + | + | 0 | 0 |

Since it had been our clinical impression that a greater and more lasting change was ensured if two or more series of treatments were given to the arthritic joints, two or more series were given in 74 patients. A review of the data revealed that if no change was observed with one series little improvement could be anticipated from further x-ray therapy. If a fair amount of improvement occurred from one treatment, slightly more effect followed a second one. The results of one and of two or more treatments are presented in Table 4. A greater improvement was noted in patients receiving two or more series. About the same number of relapses occurred, with recurrence of the inflammatory changes in the treated joint, in both groups. To date repeated courses of treatment cannot be said to guarantee permanency of the changes that take place. Any relapses usually occurred within three to six months after the completion of the series of treatments.

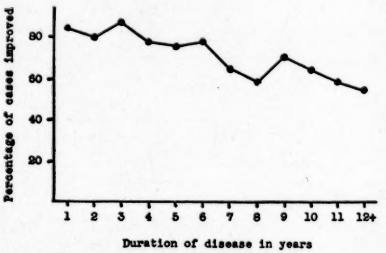


FIGURE 1. *Effect of Roentgenotherapy in Rheumatoid Arthritis in Relation to the Duration of the Disease.*

Of the total of 80 cases in which no improvement resulted from roentgenotherapy the failure of treatment was ascribed to the following factors in 33 cases of osteoarthritis: severe deformity or articular derangement in 17; no apparent cause in 9; metastatic cancer (disclosed on subsequent x-ray examination) in 4; active syphilis (found later) in 2; and active tuberculosis (subsequently discovered) in 1. In 32 patients with rheumatoid arthritis without spondylitis, the failure was associated with severe deformity or articular derangement in 18 and with no definite cause in 14, of whom 4 had severe nephritis and 2 severe cardiac disease. The failure of therapy in 15 cases of rheumatoid arthritis of the spine was associated with serious deformity and ankylosis in 7, and no cause could be determined in 8, in which severe nephritis was present in 2.

The majority of these failures, which occurred during the first three years of the study, can be attributed chiefly to a faulty selection of patients. In osteoarthritis with severe articular derangement and spur formation, little improvement can be expected from x-ray treatment. If the deformity is first corrected and the joint made mechanically as efficient as possible, roentgenotherapy hastens subsidence of the inflammation and often removes pain. Metastatic cancer is occasionally found in the presence of chronic arthritis and is often not demonstrable by x-ray for many months. Serious systemic disease and infection should not greatly hinder a local treatment like roentgenotherapy, but the experience in this study suggests that they do. The chief cause of failure, however, was some mechanical disturbance that hindered the subsidence of the articular inflammation. Although x-ray treatment has proved its usefulness in selected cases, the technic has not yet reached the stage at which the results are always predictable. The following conditions are regarded as contraindications to roentgenotherapy: quiescent rheumatoid arthritis without joint inflammation; severe deformity or articular derangement; and serious chronic disease or systemic infection.

Although no serious harm to the patients or damage to the joints was observed with the dosage given, the following reactions occurred: local pigmentation of the skin, 9 cases (2.1 per cent); nausea and vomiting, 8 spinal cases (8.1 per cent); temporary exacerbation of arthritis, 20 cases (5 per cent); amenorrhea, 2 spinal cases (9.5 per cent); and leukopenia, 1 case (0.2 per cent). It was therefore concluded that x-ray treatment must be given with caution. Pigmentation of the skin after roentgenotherapy varied tremendously in different patients, occurring to a marked degree in only 9.

Such pigmentation gradually disappeared after x-ray treatments were discontinued. Nausea and vomiting were frequent after roentgenotherapy to the lower dorsal and lumbar spine. Exacerbation of symptoms in the joint lasting from twelve to twenty-

TABLE 4. *The Effect of the Number of Courses of Treatment in Arthritis.*

| Courses of Treatment | No. of Cases | No Change | | Slight Improvement | | Moderate Improvement | | Marked Improvement | | Relapses | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | No. of cases | Per-centage | No. of cases | Per-centage | No. of cases | Per-centage | No. of cases | Per-centage | No. of cases | Per-centage |
| One | 296 | 70 | 23.6 | 69 | 23.3 | 109 | 36.8 | 48 | 16.2 | 17 | 5.7 |
| Two or more | 74 | 10 | 13.3 | 20 | 27.0 | 29 | 39.2 | 15 | 20.3 | 3 | 4.0 |

four hours after the first treatment were not unusual. Exacerbation of symptoms after subsequent treatments must be rare, since none were encountered. Among 21 women treated by roentgenotherapy for rheumatoid arthritis of the spine, amenorrhea developed in 2, one of whom was twenty-eight and the other thirty-four years of age; the condition persisted in these patients. In a fifty-six-year-old man a drop in the white-cell count from 8500 to 1800 was observed after three x-ray treatments to the spine. The patient complained of pro-

FIGURE 2. *Roentgenograms before and after Roentgenotherapy in a Fifty-Year-Old Man. The film on the left was taken before treatment. The film on the right, taken six months later, reveals no significant changes, except for a possible slight increase in osseous density.*

found weakness, and the leukopenia was an incidental finding. X-ray treatments were discontinued. Three weeks later the white-cell count returned to 7000. In no other case was any change observed in the blood. The sedimentation rate was not altered in any case, and no significant changes in the results of other laboratory tests were recorded.

## DISCUSSION

The effect of x-rays on the tissues of the joint is not fully known. It is generally assumed that the action is almost wholly local, in spite of the early uncritical assumption that systemic changes result. Some of the action is probably destructive of newly formed connective tissue, as suggested by Röhr.[13] Ewing[14] enumerated the following chief effects of x-rays on the tissues: destruction of rapidly proliferating cells; increased permeability of the cell membrane; inhibition of cell ferments; vascular changes; and lymphatic exudation. Desjardins[15] stressed the value of x-ray treatment in limiting and combating acute infections. Ochsner and Mumford,[16] without explaining its *modus operandi*, found that roentgenotherapy was helpful in relieving pain in lesions of the bones and joints. In chronic arthritis following x-ray treatment Hernaman-Johnson[17] observed local analgesia, absorption of articular exudate, decreased thickness of the articular capsule and increased density of the bone.

In recent biopsy studies following roentgenotherapy to the joints of both dogs and patients suffering from intermittent hydrops, Horwitz and Dillman[18] found a disappearance of edema from the tissues and a diffuse fibrous thickening of the vascular walls, with obliteration of the lumens of the small vascular channels. In dogs doses up to 8000 r led to loss of fur and slight superficial ulceration of the skin, but no changes in the joints. In studying the effects of x-rays on growing epiphyses, Spangler[19] and later Barr, Lingley and Gall[20] observed that severe damage and, at times, complete necrosis of the epiphyseal cartilage followed irradiation. The latter investigators also found that irradiation amounting to 1335 r or more caused epiphyseal damage in rats, but that no changes were found in the joints. Hatcher[21] reported the development of sarcoma in 3 patients given roentgen or radium irradiation; in a review of the literature he found that extremely large doses had been given whenever this complication occurred.

The patients in this study demonstrated a diminution in swelling, frequently rapid subsidence of articular exudate, extensive local analgesia and a

decrease in muscular spasm followed by an increased range of active, painless motion. In x-ray films taken two months to ten years after roentgenotherapy, no significant changes were observed in the bones. The slight changes in bone density that were found could readily be accounted for by the increased or decreased activity of the patients (Fig. 2). X-ray treatment to arthritic joints in 4 children followed four years or more caused no disturbance in growth (Fig. 3). No harmful effects on the skin or articular tissues were observed. In 2 patients in whom repeated operations permitted examination of the articular tissues before and after roentgenotherapy no significant changes were seen after a dosage of 1200 r.

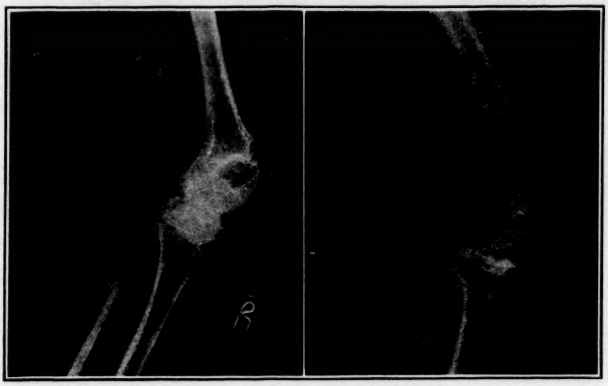

FIGURE 3. *Roentgenograms before and after Roentgenotherapy in a Five-Year-Old Girl. The film on the left, taken before treatment, shows extensive soft-tissue swelling, articular damage, and osteoporosis. That on the right, taken five years later, shows dense bones and no swelling of the soft tissues. Two courses of x-ray treatment had been given. The knee had normal painless motion.*

The various forms of chronic arthritis are usually regarded as systemic disturbances whose most obvious and severe manifestations are articular lesions. All the evidence at hand suggests that roentgenotherapy is only a local treatment to the affected joints and that its chief use is in patients in whom one or few joints show disability. The spinal joints seem to show the most rapid and definite response to the treatment, which is of little value when severe deformity or articular derangement is present. In such cases the articular inflammation may be caused mechanically by the deformity or the changes within the joint, and it may subside without other treatment when the mechanical disturbance is removed. The seriously ill patient responds slowly to roentgenotherapy — whether this is due to a profound depression of all reparative processes, we are unable to determine. The local condition of the joint is the chief indication — chronic inflammation or persistent pain and stiffness with no obvious mechanical cause. In rheumatoid arthritis of the spine, with severe bowing of the spine and ankylosis, little improvement follows roentgenotherapy.

X-ray treatment should not be used to the exclusion of other local treatments, but should be considered only a useful adjunct to the usual medical and orthopedic measures. The joints that were not treated with x-ray eventually improved for the most part, but roentgenotherapy hastened the subsidence of symptoms. All that can be expected in selected patients is an acceleration in the process of recovery. The improvement induced by x-rays is not always lasting: 56.2 per cent of patients showed improvement that persisted, without recurrence in the inflammatory process in the joint. We believe that in these cases the natural healing process has continued after improvement was started by the therapy.

The duration of the effect of x-rays apparently varies. In subsequent observation of the 370 patients only 20 showed true relapses, all of which occurred in three to six months after treatment, the average being slightly more than five months.

## SUMMARY

In this study of 370 selected patients suffering from chronic arthritis, the improvement that occurred after roentgenotherapy in 78.4 per cent was

mild in 24.1, moderate in 37.2 and marked in 17.1 per cent.

The effect varied in the various types of chronic arthritis. The greatest improvement (84.6 per cent) was seen in rheumatoid arthritis of the spine. In rheumatoid arthritis without spondylitis, 79.2 per cent of patients improved. In osteoarthritis the least improvement occurred — 72 per cent.

Suggestion apparently played little part in the results of therapy. The decrease in subjective symptoms almost paralleled the objective changes.

The duration of symptoms had little effect on the end results. The condition of the joint was the most important factor.

Repeated courses of x-ray treatment do not ensure the permanence of the improvement.

Roentgenotherapy is useful in selected cases, and its chief indications are chronic inflammation in a joint that has not responded to rest and physiotherapy, pain and stiffness in osteoarthritis, and muscular spasm and spinal limitation of motion and pain in rheumatoid arthritis of the spine.

In the 80 patients who showed no response to roentgenotherapy the principal reasons for failure were severe deformity or mechanical derangement of the joint, cancer not found in the first x-ray examination and serious systemic disease.

Unpleasant effects, such as a temporary increase in symptoms after the first treatment and nausea and vomiting, were occasionally seen, but no damage to the skin or articular tissues occurred.

Roentgenotherapy should be regarded as a local treatment only — a helpful adjunct to the medical and orthopedic measures that are usually prescribed.

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# THE DELAYED PRIMARY ATTACK OF VIVAX MALARIA*

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DURING the war, American troops stationed in areas where malaria is endemic received quinacrine (Atabrine) or quinine for suppression of malaria, the vast majority receiving the former. A great number of these men were completely free of clinical manifestations of the disease throughout the period of suppression, but after the discontinuance of suppressive medication, the first attacks of malaria occurred. Nearly all the first, or delayed primary, attacks were vivax malaria. Falciparum malaria, with few exceptions, is definitively cured by an effective suppressive regimen of quinacrine if medication is continued for three weeks or more after the last possible infection; vivax malaria is not similarly curable.

In vivax malaria, the symptoms and signs of an acute attack can be well controlled by quinacrine or quinine, but the infection is not eradicated and recurs in a great percentage of cases. The failure of the drugs to effect a complete cure of vivax infections is reflected in the occurrence of delayed primary attacks, as well as of later relapses. The delayed primary attack itself may be regarded as the recurrence of an infection acquired earlier under conditions in which the clinical manifestations were suppressed by antimalarial agents. Despite the basically similar nature of delayed primary attacks and later relapses, the former display distinctive features, which are considered in this paper.

The problem of the occurrence of delayed primary attacks in the United States will not disappear with the end of the war. With more extensive use of suppressive medication in endemic areas and with increasingly frequent travel by American residents to and from such areas, a continued, although lower, incidence of delayed primary attacks can be expected.

## MATERIAL AND METHODS

All the patients observed in this study were military personnel infected with vivax malaria of Pacific origin. Detailed observations were made in 169 cases of delayed primary attacks and in 486 cases of later relapses. The two groups were studied under the same conditions.

All patients were admitted for special treatment as soon as possible after the onset of the attack. No patient was treated unless the temperature had risen above 100°F. and a smear of the peripheral blood was positive for malaria parasites. Antimalarial therapy was begun on the morning following admission. The drugs and dosage schedules were as follows: quinacrine, 2.8 gm. in seven days (1.0 gm. the first day and 0.1 gm. three times a day for six days); chloroquine (SN 7618)[1, 2], 2.0 gm. in seven days (0.8 gm. the first day and 0.2 gm. daily for six days), 1.0 gm. in one day, 0.8 gm. in seven days (0.2 gm. the first day and 0.1 gm. daily for six days), 1.5 gm. in four days (0.6 gm. the first day and 0.3 gm. daily for three days) and 1.5 gm. in three days (0.9 gm. the first day and 0.3 gm. daily for two days); quinine sulfate, 29.0 gm. in fourteen days (3.0 gm. the first day and 2.0 gm. daily for thirteen days); and quinine-plasmochin, 29.0 gm. of quinine and 0.84 gm. of plasmochin naphthoate in fourteen days (the former according to previous schedule, and the latter, 0.02 gm. every eight hours). The ratio of delayed primary attacks to later relapses was essentially similar in the various treatment groups.

Daily observations were made of the symptoms of the attack, evidences of toxicity of the drugs used and physical findings.

Counts of malaria parasites in the peripheral blood were done twice daily, in the morning and in the afternoon, until a negative smear was obtained, after which three consecutive negative daily smears were required.† The great majority of parasite counts were made by the Earle–Perez[3] method, which utilizes a thick blood smear; the rest of the counts were determined by the leukocyte-parasite ratio, in which a white-cell count is performed and the parasite count calculated according to the ratio of parasites to white cells in a thin blood smear. The two procedures give similar results, although the former is much simpler.

After the completion of treatment, most of these patients were transferred to the convalescent section of the hospital, where they were observed until relapse or for a period of one hundred and twenty days from the last day of treatment. No antimalarial drugs were given during this period, and smears were examined twice weekly. The occurrence of a positive smear in association with a temperature above 100°F. was considered a clinical relapse, and such patients were readmitted to the treatment ward for further study and therapy. A positive smear without fever or symptoms was regarded as a parasitemic relapse and in these cases temperature observations were made three times daily and smears were examined daily until negative; if fever supervened, the patient was admitted to the ward with a diagnosis of clinical relapse.

## CLINICAL CHARACTERISTICS

Most delayed primary attacks are clinically indistinguishable from later relapses of vivax malaria.

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†All counts were performed by the Imported Malaria Studies Laboratory, United States Public Health Service, under the direction of Captain Don E. Eyles.

The characteristic febrile paroxysms, with associated symptoms and physical findings, occur in delayed primary as well as in recurrent attacks. In Table 1 the incidence of these symptoms and signs is compared in two groups of patients, one of delayed primary attacks and the other of later relapses. Both groups were observed under the same conditions and were treated with the same drug regimens. It is readily apparent that the two groups showed little difference in the incidence of symptoms and physical findings.

Occasionally, however, the recognition of malaria in a delayed primary attack represents a difficult diagnostic problem. In some cases, smears are persistently negative for several days, despite the occurrence of chills and fever and other commonly associated symptoms. Such cases are often differentiated with difficulty from a variety of bacteremias or septicemias. The presence of leukopenia in malaria is of some help in ruling out streptococcal, pneumococcal or staphylococcal bacteremias, but does not differentiate malaria from typhoid fever, dengue or cases of influenza in which respiratory symptoms are minimal.

TABLE 1. *Incidence of Symptoms and Physical Findings in 84 Delayed Primary Attacks and 190 Later Relapses.*

| Symptom or Physical Finding | Percentage of Delayed Primary Attacks | Percentage of Later Relapses |
|---|---|---|
| Chilly sensation | 98 | 95 |
| Chills | 93 | 90 |
| Headache | 96 | 97 |
| Backache | 89 | 93 |
| Generalized aching | 91 | 93 |
| Weakness | 96 | 97 |
| Nausea | 52 | 71 |
| Vomiting | 36 | 49 |
| Dizziness or lightheadedness | 27 | 37 |
| Tinnitus | 36 | 45 |
| Abdominal pain | 61 | 65 |
| Abdominal tenderness | 60 | 65 |
| Palpable spleen | 69 | 71 |
| Palpable liver | 52 | 48 |
| Herpes simplex | 27 | 21 |

Some delayed primary attacks are ushered in by an irregular fever, without chills and with negative malaria smears. In some cases the fever eventually assumes a tertian character, providing a clue to the diagnosis; in others a chill occurs after a few days of remittent fever and is followed by a more intermittent febrile course.

The onset of a delayed primary attack may be marked by severe abdominal pain and tenderness, and by nausea and vomiting. When these symptoms occur before a paroxysm supervenes, they simulate closely an acute abdominal condition, but the presence of a low white-cell count is a guide to the correct diagnosis.

All these variants from the typical vivax attack occur in later relapses, as well as in the delayed primary attack. The absence of a previous history of malaria, however, and the greater incidence of negative smears or of extremely low parasite counts at the onset of an attack render the establishment of a diagnosis much more difficult in the delayed primary than in the later attacks. When clues to the correct diagnosis are furnished by a history of residence in an endemic area, the discontinuance of suppressive medication within the previous three or four months and enlargement of the spleen or liver, thick blood smears should be examined at least twice daily until the diagnosis is established.

When faced with fever, chills and severe malaise in a patient who exhibits no diagnostic physical findings and has several negative malaria smears, the physician may be tempted to employ sulfonamides or penicillin or to attempt a therapeutic trial with an antimalarial drug. Such temptation should be resisted. Penicillin is of no value in the treatment of malaria, and the sulfonamides and antimalarial drugs can prevent the appearance of malaria parasites and thus make impossible the establishment of a correct diagnosis. It is essential that the diagnosis of malaria be verified by positive blood smears, so that rational treatment can be instituted for the current attack and subsequent attacks can be promptly recognized and properly treated.

## Interval between Cessation of Suppressive Medication and Onset of Delayed Primary Attack

Information on 101 patients with delayed primary attacks was assembled, to calculate the interval between the time of discontinuance of suppressive medication and the date of onset of the first attack. In 96 cases data on the dosage of quinacrine taken for suppression were available. For purposes of analysis, the patients were divided into three groups — those who had taken the usual 0.7 gm. of quinacrine during each week, those who had taken more than 0.7 gm. per week, and those who had taken less than 0.7 gm. per week (Table 2).

It is clear that despite considerable variations in the suppressive regimens used in the endemic areas, the mean interval to onset of the first attack following the end of suppression in each of the three groups was essentially the same — forty-one days. The range of intervals was also similar in each of the groups, and the standard deviation was nearly identical: 23.3 in the first, 22.9 in the second and 21.3 in the third. Accordingly, one can predict that two thirds of patients who develop delayed primary attacks will do so nineteen to sixty-three days following the discontinuance of suppressive medication. It must be appreciated, however, that in rare cases the delayed primary attack occurs as long as one year after discontinuance of quinacrine suppression.

## Initial Parasitemia

It was pointed out above that malaria smears are frequently negative at the onset of symptoms in

delayed primary attacks. This observation indicated that some patients in the primary attack are unduly sensitive to the malaria parasite and have a marked clinical reaction to low parasitemic levels. This occurrence is more frequent in the delayed primary attack than in relapses. An analysis was made of the parasite counts taken in the morning of the first day of treatment prior to the administration of the first dose of drug. The frequency distribution of initial parasite densities in delayed primary attacks and relapses is presented in Figure 1.

TABLE 2. *Interval Between End of Suppression and Onset of Delayed Primary Attack.*

| No. of Patients | Suppressive Quinacrine Dosage | Range of Interval | Mean Interval |
|---|---|---|---|
| | *gm. per week* | *days* | *days* |
| 19 | Less than 0.7 | 3 to 87 | 41.5 |
| 55 | 0.7 | 8 to 104 | 42.2 |
| 21 | More than 0.7 | 10 to 83 | 39.9 |

It is evident that the level of parasitemia at the onset of an attack is significantly lower in the delayed primary attack than that in the later relapses. Fifty-four per cent of the delayed primary cases had counts less than 1000 per cubic millimeter, whereas this was true in only 31 per cent of later relapses. These findings are in agreement with the clinical impression that the majority of patients in the former group show a greater symptomatic reaction to low parasite concentrations than those in the latter. Low parasite concentrations in primary attacks were observed by Eyles and Young,[4] who reported counts of less than 1000 per cubic millimeter at the onset in 47 of 50 patients in whom Pacific vivax malaria was induced by means of infected mosquitoes. A large proportion of patients with a parasite density of less than 1000 per cubic millimeter have thin smears that are completely negative or show extremely rare parasites. These findings indicate the importance of thick smears for the detection of malaria, particularly in the primary attack.

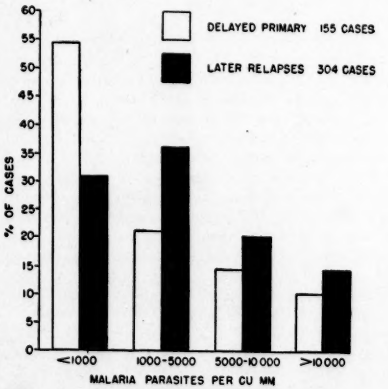


FIGURE 1. *Initial Parasite Densities in Delayed Primary Attacks and Relapses of Pacific Vivax Malaria.*

## Parasite Clearance Rate

It has been our experience in the treatment of large numbers of cases of vivax malaria that, on the whole, patients with low parasite counts at the beginning of treatment are more rapidly cleared of parasitemia than those with high initial parasite counts. Inasmuch as the initial parasitemia is significantly lower in the delayed primary attacks than in the later relapses, one might expect that cases in the former group would be more rapidly cleared of parasites than those in the latter; the opposite, however, was observed.

Table 3 presents a comparison of the rate of parasite clearance in delayed primary attacks and in later relapses. The ratio of delayed primary attacks to later relapses was essentially similar in all treatment groups and offset known variations in the rates of parasite clearance by quinine, quinacrine, chloroquine or plasmochin. Twenty-four hours after the beginning of treatment, only 17 per cent of delayed primary cases had negative smears, whereas 25 per cent of later relapses were negative. This difference is statistically significant (probability 0.029). On subsequent smears the rates of clearance were gradually equalized.

TABLE 3. *Parasite Clearance in Treatment of Delayed Primary Attacks and Relapses.*

| Clearance after First Dose of Drug | Delayed Primary Attack (152 cases) | Later Relapses (486 cases) |
|---|---|---|
| *hr.* | % | % |
| 24 | 17 | 25 |
| 48 | 67 | 73 |
| 72 | 91 | 94 |
| 96 | 99 | 100 |
| 132 | 100 | |

## Control of Fever

The temperature course is a valuable index of the response of a malaria patient to therapy. The presence or absence of fever on the second day of treatment was found to be a useful measure of this response.

This measure was applied to the cases of delayed primary attack and to those of later relapse in the same treatment groups in which the rates of parasite

clearance were studied. Patients with an oral temperature above 100°F. were regarded as having fever. Cases in which a temperature above 100°F. represented merely the defervescence from the peak of a paroxysm on the first day of treatment were not included. An analysis of the occurrence of fever on the second day of treatment in 150 cases of delayed primary attack and 465 cases of later relapse revealed a significantly higher incidence in the former (27 cases, or 18 per cent) than in the latter (29 cases, or 6 per cent). This higher incidence may have been related to the slower parasite-clearance rate in the delayed primary attacks, as suggested by the fact that of the total of 56 patients with fever on the second day only 4 per cent were found to have negative smears on that day, whereas of 559 patients who were afebrile on the second day, 25 per cent had negative smears.

As measured by the control of fever and parasitemia, the delayed primary attack responded less readily to effective antimalarial therapy than the later relapse.

### Relapse Rate and Interval to Relapse

The efficacy of antimalarial therapy is measured not only by its control of the acute attack but also by its effect on the incidence of subsequent-relapse and on the length of interval to relapse.

After treatment with one of the antimalarial drug regimens, each patient was observed until the next attack or, if no attack occurred, for one hundred and twenty days after the last day of treatment. The rate and interval to relapse within one hundred and twenty days could thus be established for each group. In view of the differences noted between the response to treatment of acute attacks in delayed primary attacks and that in later relapses, it was of interest to determine whether differences also existed in the relapse rate and the interval to relapse.

The results of such an analysis in cases followed until subsequent relapse or for one hundred and twenty days are presented in Table 4, which does not include patients treated with combined quinine and plasmochin, in whom the incidence of relapse within one hundred and twenty days is less than 10 per cent.[5] The rates of relapse and the mean intervals to relapse were nearly identical in both groups, as was the distribution of intervals to relapse within the observation period of one hundred and twenty days.

Table 4. *Relapse Rate and Mean Interval to Relapse Following Treatment of Delayed Primary Attacks and Relapses.*

| Type of Attack | No. of Cases | Clinical Relapse within 120 Days | | Mean Interval to Relapse |
|---|---|---|---|---|
| | | No. of cases | Per-centage | |
| | | | | *days* |
| Delayed primary attack | 109 | 76 | 69.7 | 54.4 |
| Later relapse ..... | 259 | 193 | 74.6 | 53.2 |

### Discussion

The observations presented above indicate that repeated attacks of malaria produce a somewhat altered response to treatment of an acute attack, patients with later relapses showing a diminished sensitivity to the malaria parasite as demonstrated by the higher level of parasite density required to produce symptoms. The greater effectiveness of antimalarial agents in the control of fever and in the clearance of parasites in the later relapses is a further indication that some immunity is developed during the course of recurrent attacks. Such immunity, however, is sufficient neither to diminish the likelihood of a recurrence within one hundred and twenty days after treatment of an attack nor to cause a lengthening of the interval between relapses.

The data on the interval to the delayed primary attack following the end of suppressive medication raise some interesting questions. As pointed out above, the mean interval to the first attack in 101 cases was forty-one days, with a range of three to one hundred and four days. This wide range obtained in patients who received large doses of suppressive quinacrine as well as in those treated with the usual dosage of quinacrine. An adequate concentration of quinacrine in the plasma is necessary to maintain effective suppression of the symptoms of malaria. There is a considerable range in the plasma concentrations of quinacrine achieved on suppressive regimens, and there are corresponding variations in the time required for the concentration to drop to its minimal effective level after the cessation of medication. Such factors probably account for some of the variation in length of intervals to primary attacks, but not for the wide range of intervals observed. Symptoms do not necessarily develop as soon as the quinacrine concentration in the plasma has fallen below the minimal effective level. One of the other factors that possibly determine the interval may be the natural periodicity of recurrent vivax infections.

Many patients who had had several attacks of malaria stated that the attacks recurred at regular intervals, usually every six to eight weeks, and many followed through the course of several recurrences did show such periodicity. If one assumes a normal variation among patients in the length of the periods between attacks and differences in the periodicity of various strains of vivax parasites, the wide range in the intervals to delayed primary attacks becomes understandable.

During the course of suppression there may be recurrent episodes of malarial activity in which the clinical manifestations are not evident so long as the plasma concentration of quinacrine remains at an effective level. When suppression is discontinued

and the plasma concentration declines below the effective level, a clinical attack becomes possible. But the time of occurrence may depend on the periodicity of the infection in individual cases and the relation of the last preceding episode of suppressed malarial activity to the date of the last effective quinacrine concentration in the plasma. Thus, if a period of suppressed activity occurs immediately before the end of suppression, the delayed primary attack usually does not follow for another six to eight weeks. If the periodicity is longer, the attack may not occur until as long as one hundred days later. In cases in which the interval to the first attack is even longer, it is possible that one or more periods of subclinical malarial activity occur before a full-blown clinical attack develops. If a period of activity is due immediately after the discontinuance of suppressive medication in a patient who is highly sensitive to the malaria parasite, the delayed primary attack may readily occur within the first week after the end of suppression.

These considerations, although speculative, seem best to explain the wide range of intervals to the first attack observed following cessation of suppression.

## Summary

In this study, based on 169 cases of delayed primary attacks and 486 later relapses occurring in military personnel with vivax malaria of Pacific origin, most of the delayed primary attacks were clinically indistinguishable from later relapses. Occasionally, however, such attacks posed problems in diagnosis. Treatment with sulfonamides, penicillin or antimalarial drugs should be withheld until a diagnosis is established. The existence of malaria should be proved by positive blood smears, so that rational treatment can be instituted and subsequent malaria attacks can be promptly recognized and properly treated.

The mean interval between the cessation of suppression and the onset of the delayed primary attack in 101 cases was forty-one days. One can predict that two thirds of delayed primary attacks will occur between approximately the third and the ninth week after the end of suppression.

The parasite concentrations in the peripheral blood were significantly lower at the onset of delayed primary cases than those in later relapses. Accordingly, thick-smear examinations should be made at least twice daily until parasites are found.

The rate of parasite clearance under treatment was somewhat slower in the delayed primary attack than in the later relapse, despite the lower initial parasite counts.

Fever was less readily controlled in the delayed primary attack than in later recurrences.

The incidence of clinical recurrence within one hundred and twenty days after treatment of the acute attack was essentially the same for delayed primary cases and later relapses. The mean interval to relapse was also nearly identical in both groups, as was the distribution of intervals to relapse within the observation period of one hundred and twenty days.

The higher initial parasitemia in later relapses suggests a diminished sensitivity to the malaria parasite. This finding, in addition to the more rapid parasite clearance and control of fever that occur in the treatment of later relapses, indicates the development of some immunity in the course of recurrent vivax malaria infection.

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# QUARTAN MALARIA OCCURRING SUBSEQUENT TO A BLOOD TRANSFUSION*

## Report of a Case

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WITH the return of military personnel from areas where malaria is endemic, the problem of post-transfusion malaria is bound to assume greater importance than it has in the past. The following case deserves mention because of several unusual features.

### CASE REPORT

E. M., a 40-year-old married woman, was admitted to the Rhode Island Hospital on February 25, 1946, complaining of chills and fever every 3rd day for the last several weeks. She had previously been seen at the hospital on September 19, 1945, when she was admitted in shock. At that time she gave a history of vaginal bleeding for 1 week and profuse bleeding for 3 days following self-attempted uterine instrumentation. While in the hospital she received three 500-cc. transfusions of stored blood and 1 unit of plasma. With penicillin and supportive treatment she made an uneventful recovery and was discharged on the 10th hospital day. Following her return home she had considerable irregular vaginal bleeding, but she gained weight and strength and was able to do her housework adequately. At about 7:00 p.m. on November 23, after a short period of malaise, she suddenly experienced a severe shaking chill, which lasted about 2 hours. This was followed by severe drenching sweat, backache and headache, which had entirely disappeared by the following morning. On November 26 malaise and a chill, followed by sweating, backache and headache, returned. She was seen by a physician, who was aware of the cause of her recent hospitalization and who gave her one of the sulfonamides — it was estimated that she took about 7 gm. daily for 4 days. During the month of December, there was only 1 episode of chill and fever, which occurred about the middle of the month. The febrile episodes began again on January 12 and occurred at irregular intervals throughout the month. The patient had no symptoms during the first week in February but on February 8 there was a recurrence of chills and fever. From that time until 2 days before admission to the hospital, the chills and fever occurred regularly every third day at a time that the patient was able to predict.

There was no family or past history of malaria, tuberculosis, diabetes or cancer. The patient was born in Vermont, where she had lived for about 37 years before moving to Providence, and had never been outside the New England states. Her health had always been good, except for scarlet fever in childhood and an appendectomy and right salpingo-oophorectomy at the age of 14. She had had twelve pregnancies, 8 babies having been delivered at term, with four pregnancies terminated by induced abortion.

Physical examination revealed a rather thin but well developed woman who appeared well. The temperature, pulse and respirations were normal. The blood pressure was 110/70 in each arm. The examination was entirely negative, except that the oral mucous membrane appeared slightly pale. The liver was not palpable and was percussed at the costal margin on deep inspiration. The spleen was not felt.

On the day of admission, x-ray examination of the chest was reported as negative, and the urine was said to contain a trace of albumin and a trace of sugar.

At about 1:00 p.m. on the 2nd hospital day, just as the patient had predicted, she began to have malaise, backache and headache. These symptoms progressed, and the temperature rose; about 2 hours later she was at the height of a moderately severe paroxysm, with an oral temperature of 105°F. A thick blood film, which was taken during the paroxysm and reported as negative for malarial parasites, was later reviewed and showed quartan parasites, *Plasmodium malariae*. Examination of the blood showed a red-cell count of 3,700,000, with a hemoglobin of 12.6 gm., and a white-cell count of 5950, with 74 per cent neutrophils, 20 per cent lymphocytes and 6 per cent monocytes. The parasite density during the paroxysm, as determined from the original blood film by the method of Watson,[1] was 60 organisms per cubic millimeter. The blood urea nitrogen was 7 mg. and the blood sugar 77 mg. per 100 cc. The blood Hinton reaction and agglutinin tests for typhoid, undulant and typhus fever were negative. A blood culture was sterile. An electrocardiogram was within normal limits. By 7:00 p.m. the patient was much improved, and by the next morning, except for slight weakness, she was entirely free of symptoms.

At 8:30 p.m. on the 4th hospital day blood films revealed a parasite density of 1566 organisms per cubic millimeter. The white-cell count was 4450. Earlier in the day, an intravenous pyelogram had been reported as normal. At 9:30 a.m. on the 5th hospital day the parasite density was 576 organisms per cubic millimeter, and examination of the blood showed a red-cell count of 4,270,000, with a hemoglobin of 11.3 gm., and a white-cell count of 5450, with 48 per cent neutrophils, 49 per cent lymphocytes and 3 per cent monocytes. The urine showed a trace of albumin but no bile and a urobilinogen concentration of 0.65 mg. per 100 cc. The blood urea nitrogen was 8 mg., the blood sugar 77 mg., and the blood cholesterol 118 mg. per 100 cc., and the icteric index was 4.2.

As expected, in the afternoon, the patient experienced a malarial paroxysm almost identical to the previous one. At what was judged to be the height of this episode the parasite count was 410 organisms per cubic millimeter. Examination of the blood showed a red-cell count of 4,260,000 and a white-cell count of 4300 with 53 per cent neutrophils, 46 per cent lymphocytes and 1 per cent monocytes. At that time a blood volume determination was done with the dye T-1824,§ the 10-minute sample method of Gregersen,[2] as modified to the use of the Cencophotelometer, being used. The dye was injected by means of a calibrated syringe. Hematocrit determinations on venous blood were done in Wintrobe tubes, and plasma protein determinations by the method of Bowman[3] were performed on the control and dye samples. These determinations were repeated on the 11th hospital day, after 6 days with no fever or symptoms (Table 1). Quinacrine, 0.2 gm. by mouth, was begun at 4:00 p.m. It was planned to continue this dosage every 6 hours for five doses for a total of 1 gm. The patient received 1.4 gm. in 0.2-gm. doses in the succeeding 48 hours. The dosage then was changed to 0.1 gm. three times daily for five more days. The total dosage was 2.9 gm.

At 9:00 a.m. on the sixth hospital day, the parasite density was 432 organisms per cubic millimeter, and examination of the blood showed a red-cell count of 3,950,000, with a hemoglobin of 11.5 gm., and a white-cell count of 3600, with 57 per cent neutrophils, 29 per cent lymphocytes, 13 per cent monocytes and 1 per cent eosinophils. The urine contained no bile, and the urobilinogen concentration was 0.4 mg. per 100 cc.

At 10:00 a.m. on the 7th hospital day the parasite density was 450 organisms per cubic millimeter, and the white-cell count was 4500.

On the 8th hospital day — the 4th day of quinacrine treatment — no parasites were seen in the thick blood film. Examination of the blood showed a red-cell count of 3,910,000 with a hemoglobin of 11.5 gm., and a white-cell count of 5500; no differential count was done. On the 10th hospital day examination of the blood showed a red-cell count of 4,150,000, with a hemoglobin of 11.5 gm., and a white-cell count of 4200, with 63 per cent neutrophils, 32 per cent lymphocytes, 4 per cent monocytes, and 1 per cent eosino-

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phils. On the 11th hospital day no parasites were seen in the thick blood film. The blood cholesterol was 140 mg. per 100 cc., and the icteric index was 3.3. The urine showed a concentration of urobilinogen of 0.1 mg. per 100 cc.

At no time during the hospital stay was the spleen palpable, and only once was the liver edge felt at the costal margin on deep inspiration. The patient was discharged on the 14th hospital day, and when seen in follow-up on April 6, she stated that she had had no further symptoms of malaria.

Figure 1 shows the relations of the fever and pulse rate to the parasite densities and the white-cell count and the response to quinacrine treatment.

Rubenstein, Shulman and Merrill[4] reported 12 cases of malaria occurring subsequent to blood transfusion in Massachusetts since 1929. Six cases were reported during the years 1939 to 1944, and at least 9 — possibly 10 — of the cases were quartan in type. Ten of the 12 donors involved in the study had lived for a period of years in a country where malaria is known to be endemic. In the other 2 cases information concerning the donor's country of residence was not available. The authors regarded the length of time of residence in a new country where insect transmission does not exist or is exceedingly rare as the known period of latency of the malarial infection, whether or not the donor presents a history of malaria. In the 5 donors in whom the duration of latency of the malarial infection was established, it varied from twelve to twenty-seven years. Not one of these donors had a history of manifest malaria. The duration of latency of infection in 3 donors with a history of malaria could not be stated. One of the 3 had a positive blood film for malaria. The other positive blood film was found in a patient with no history of malaria. In 8 of the cases of quartan malaria reported by these authors, the incubation period varied from twenty-one to one hundred and eleven days, with an average of fifty-two days.

McClure and Lam[5] reported 2 cases of quartan malaria subsequent to the transfusion of blood that had been stored, in each case, for five days. Both donors had been born and had spent their childhood in Sicily. One, who gave no history of malaria, had

TABLE 1. *Blood-Volume Determinations with T-1824.*

| Hospital Day | Temperature | Hematocrit | | Plasma Protein | | Blood Volume | | Plasma Volume | | Erythrocyte Mass | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Measure | Change | Measure | Change | Measure | Change | Measure | Change | Measure | Change |
| | °F. | % | % | gm. per 100 cc. | % | cc. | % | cc. | % | cc. | % |
| Fifth | 105 | 37.4 | | 6.4 | | 4630 | | 2900 | | 1730 | |
| Eleventh | 98.6 | 38.6 | +3 | 6.7 | +5 | 4500 | -3 | 2754 | -5 | 1746 | +1 |

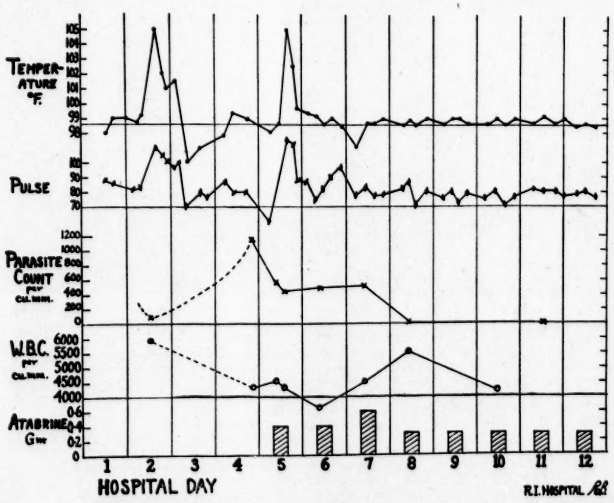


FIGURE 1. *Relation of Clinical Course to Parasite Densities, White-Cell Count and Treatment with Quinacrine.*

lived in the United States for thirty-two years. The other, who gave a history of malaria in childhood, had lived in Michigan for thirty-six years. Blood smears were examined for malaria on one occasion in both donors, but no parasites were seen.

Sharnoff, Geiger and Seltzer[6] reported 2 cases of malaria following the transfusion of stored blood. One of them was the quartan type. The 3 donors involved in this case were natives of New Jersey, the British West Indies and Puerto Rico, respectively. Blood samples from these donors were stored eight, two and eight days, respectively. The other case was tertian (vivax) malaria. The donor involved was a Puerto Rican, and the blood had been stored for two days.

With the exception of the case presented above, no cases of malaria subsequent to blood transfusion have been reported from the blood bank of the Rhode Island Hospital, which from May, 1942, to January 1, 1946, supplied blood for about forty-five hundred transfusions.[7]

Of the 3 donors involved in this case, 2 were Italian born. One was a fifty-six-year-old native of Naples, who gave a history of malaria that had been treated with quinine forty years previously. He had subsequently been free of the symptoms of malaria and had lived in New England ever since his immigration thirty years previously. The second donor, a fifty-seven-year-old native of Brindisi, gave a history of malaria treated successfully with quinine forty-six years previously and stated that he had been free of the symptoms of malaria ever since that time and had lived in New England since his immigration ten years later. The third donor, a fifty-five-year-old native of Providence, stated that between the ages of six and thirteen years she had had malaria every summer. These attacks had all been treated with quinine and had not recurred in the last forty-two years.

Thick blood films were examined once in all 3 donors, and twice in 1, but no malarial parasites were seen. The blood used for the transfusions had been stored at a temperature of 6°C. for five days in the first donor, seven in the second, and twelve in the third. In this reported case the length of time between the transfusions of blood and the appearance of the clinical symptoms of malaria was sixty-six days.

In the clinical study of this case, two features were of especial interest. The first was that low parasite densities were maintained during the period of study (Fig. 1). Kitchen[8] states that *P. malariae* appears to be more toxic, in relation to parasite density attained, than either *P. vivax* or *P. falciparum*. The second feature was that blood-volume determinations, one made during a paroxysm and another in convalescence six days later, showed no significant alterations in the plasma, blood or circulating erythrocyte volumes (Table 1). Feldman and Murphy[9] described blood, plasma and erythrocyte changes occurring in infections with *P. vivax* and *P. falciparum* that reflect the response of the body to erythrocyte destruction. The absence of significant erythrocyte destruction, owing to the low parasite densities attained, may explain the absence of significant blood-volume changes in the case reported.

## Summary

A case of quartan malaria, caused by *Plasmodium malariae*, in a native of New England subsequent to the transfusion of stored blood is reported. The three donors involved gave past histories of malaria forty, thirty-six and forty-two years previously, but blood films failed to reveal malarial parasites.

The course of the illness, the parasite densities attained, the white-cell counts and the response to quinacrine therapy are discussed.

Plasma, total blood and erythrocyte volumes, determined with the dye T-1824, were essentially the same during a paroxysm as during convalescence.

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# PELVIC INFLAMMATORY DISEASE*

## A Survey of 300 Consecutive Cases, with Special Reference to Treatment

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THE sulfonamides have become well established as invaluable agents in the treatment of many infective conditions. As a result, the tendency has been to regard the drugs equally effective in any infective state, and they are given routinely by many physicians whenever a febrile inflammatory condition develops.

For some time, we had doubted the value of the sulfonamides in many of the cases of pelvic inflammatory disease under our care at the Metropolitan Hospital. We therefore examined the literature to determine the experience of others in the use of the drugs in such diseases.

As early as 1938, Gillett[1] believed that sulfanilamide was 100 per cent effective in the treatment of acute salpingo-oophoritis without demonstrable inflammatory masses and in recurrent acute salpingo-oophoritis. He conceded, however, that there was little, if any, improvement if adnexal masses were present.

In 1938, Goff[2] stated that vigorous sulfanilamide therapy within the first two or three days of an acute gonorrheal infection completely aborted the process in most cases, but that a permanent cure did not necessarily follow. He reported that in patients seen late in the course of infection an occasional pelvis cleared up but that in most cases some residual infection remained. He believed that probably 50 per cent of cases eventually required operation — an operative incidence much higher than that in the cases in this study.

In 1941 Williams[3] compared the effect of sulfanilamide therapy on 72 patients with acute pelvic inflammatory disease with that obtained in 100 patients treated similarly except that the drug was not given. He concluded that the dramatic results often obtained with sulfanilamide in streptococcal infections and in pneumonia were disappointingly absent. He stated that, if given early, the drug prevented the development of tubal involvement in gonorrheal infection but was of little value once the inflammatory process in the adnexa had become well established.

Barrows and Labate,[4] in 1943, studied 204 cases of acute salpingitis treated with sulfanilamide and sulfathiazole, of which 71 were primary and 133 acute exacerbations. They found that primary attacks of less than five days' duration were apparently cured after a week of chemotherapy in 70 per cent of the mild and 66 per cent of the moderate cases, with resolution of adnexal masses. Mild and severe cases of longer duration and recurrent cases showed no adequate response.

In a study of pelvic inflammatory disease of specific origin, Miller[5] asserted that, except for general measures, no treatment of pelvic inflammatory disease gives satisfactory results, chemotherapy being disappointing in infections above the internal cervical os. He also stated that surgery is ultimately necessary in a large proportion of cases of recurrent salpingitis, particularly in public institutions with a large number of Negro patients, and that conservative surgery must frequently be followed by secondary surgery. Acute appendicitis furnished the chief difficulty in differential diagnosis. He concluded that bacteriologic diagnosis, whether by smear or culture, is highly unsatisfactory.

It was thus evident that our opinion was — at least in part — shared by others, and a survey of our own cases at the Metropolitan Hospital was undertaken. It was decided to analyze a large number of consecutive cases of pelvic inflammatory disease without attempting to classify the type of disease or the cause. The patients had two things in common: all had inflammatory disease of the pelvis and all were ill enough to be hospitalized. Some were desperately ill, and 2 died. Some of these patients were treated by bed rest alone, others by bed rest and sulfonamides and still others by bed rest and surgery.

### GENERAL CONSIDERATIONS

The youngest patient in the series was thirteen and the oldest fifty-two years of age. One hundred and sixty-seven patients (56 per cent) were under twenty-five, 90 (30 per cent) were between twenty-five and thirty-five, and only 43 (14 per cent) were over thirty-five.

Two hundred and sixteen patients (72 per cent) were Negresses. This was chiefly due to the fact that the district served by the hospital is largely populated by Negroes, but other factors, such as social and economic status, played a definite role. Because of this racial distribution, most of the patients in this series were probably more neglected than the average patient, with a higher morbidity, mortality and need for operation.

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As might be expected previous gravidity was more frequent in the older than in the younger patients. Of those over thirty-five years of age 84 per cent had been gravid, and of those between twenty-five and thirty-five 78 per cent had been gravid, whereas of those under twenty-five only 54 per cent had been gravid. This is one of the reasons for the policy of conservatism in the younger group whenever possible.

All patients were kept in bed and received routine nursing care, with the occasional use of codeine and aspirin for relief of pain. Patients who received sulfonamide therapy were given sulfanilamide or sulfadiazine, or both. The daily dosage varied from 3 to 4 gm., along with an alkali — usually sodium bicarbonate — in suitable dosage.

If one assumes that 25 mm. in one hour by the Westergren technic is the normal upper limit of the blood sedmentation rate, 131 cases (44 per cent) were above and 166 cases (56 per cent) were below this level on admission to the hospital.

The chief complaints in the entire series were as follows: pelvic pain, 95 per cent; vaginal discharge, 33 per cent; bleeding, 10 per cent; urinary frequency, 10 per cent; and backache, 10 per cent.

To our knowledge, three errors in diagnosis were made, all in cases diagnosed as acute appendicitis that were in fact pelvic inflammatory disease.

The indications for operation in these cases were persistence of masses or of subjective symptoms or both. A prerequisite for operative interference in most cases was a sedimentation rate of 25 mm. or less, to give assurance that active infection was not present.

## Results of Treatment

The results of the various forms of treatment are presented in Table 1.

In classifying the cases, improvement consisted in relief of symptoms or subsidence of the disease, or both. Operative cases in which any portion of the adnexa remained after conservative surgery were regarded as improved rather than cured, owing to the potentiality of involvement of the remaining adnexa; cure in such cases implied complete removal of adnexa, including all the diseased tissue, and complete relief of symptomatology.

Table 1. *Results of Treatment.*

| Treatment | Age Group yr. | No. of Cases | Hospital Stay days | No Improvement | Improvement | Cure | Death |
|---|---|---|---|---|---|---|---|
| Bed rest alone | Under 25 | 66 | 13.3 | 1 | 65 | 0 | 0 |
| | 25-35 | 34 | 13.4 | 2 | 27 | 5 | 0 |
| | Over 35 | 8 | 21.0 | 0 | 6 | 2 | 0 |
| Totals | - | 108 | | 3 (3%) | 98 (91%) | 7 (6%) | 0 |
| Average | | | 15.9 | | | | |
| Bed rest and sulfonamides | Under 25 | 93 | 13.8 | 3 | 86 | 4 | 0 |
| | 25-35 | 36 | 15.8 | 1 | 29 | 4 | 2 |
| | Over 35 | 19 | 16.1 | 1 | 17 | 1 | 0 |
| Totals | - | 148 | | 5 (3%) | 132 (89%) | 9 (6%) | 2 (1%) |
| Average | | | 15.2 | | | | |
| Bed rest and surgery | Under 25 | 7 | 19.4 | 0 | 5 | 2 | 0 |
| | 25-35 | 19 | 24.5 | 0 | 13 | 6 | 0 |
| | Over 35 | 15 | 21.9 | 0 | 8 | 7 | 0 |
| Totals | - | 41 | | 0 | 26 (63%) | 15 (37%) | 0 |
| Average | | | 21.9 | | | | |

In unoperated cases, cure consisted in complete resolution of all obvious disease and disappearance of all symptoms.

Experience indicated that many patients regarded as improved later had recurrences of the disease.

The following are brief summaries of the 2 fatal cases:

Case 1. A 26-year-old Negress who had had four pregnancies and given birth to 2 children was admitted to the hospital with acute pelvic inflammatory disease and peritonitis. The sedimentation rate varied from 25 to 100 mm. The patient was given transfusions, infusions, sulfathiazole and sulfadiazine. There was an elevation in temperature daily up to 103°F. The patient progressively failed, dying on the 22nd hospital day of peritonitis.

Case 2. A 26-year-old Negro primipara was admitted to the hospital with septic, incomplete abortion, parametritis and thrombophlebitis of the pelvic veins. The uterus was evacuated with sponge forceps. Seven small blood transfusions, as well as sulfathiazole and sulfanilamide were given. The patient died on the 73rd hospital day of septic pulmonary emboli and abscess formation.

An interesting observation is that over four fifths of the cases in this series of pelvic inflammatory disease occurred in women under thirty-five and more than half in women under twenty-five years of age. This fact suggests that such disease is characteristic of the early part of sexual life.

The use of sulfonamides appeared to have no influence on the average period of hospital stay, which in patients treated with bed rest alone was almost identical with that in patients treated with

bed rest and sulfonamides. Operative treatment lengthened the hospital stay, but this is understandable, in view of the fact that most patients with high sedimentation rates were kept in bed until this evidence of acute inflammation had subsided. The time required for such a lowering of the sedimentation rate varied but was usually a week or ten days. The duration of hospital stay in these cases was on the average eight days longer than that in the cases without operation.

Improvement obtained with bed rest alone when compared with that afforded by bed rest and sulfonamides showed little difference — 91 per cent in the former and 89 per cent in the latter. The small number of patients regarded as cured is outstanding evidence of the inadequacy of these methods of treatment. Cure was obtained in 6 per cent of cases both with bed rest alone and with bed rest and sulfonamides. The percentage of cures in the operative cases was higher, sixty-three per cent of patients being improved and 37 per cent cured. There were no deaths in the operative series.


### Summary

A group of 297 patients with pelvic inflammatory disease was studied. The incidence of disease was much greater in the younger than in the older age group, decreasing progressively with age. Pelvic pain and discharge constituted the most frequent chief complaints.

The therapeutic results obtained with bed rest alone and with bed rest with the administration of sulfonamides were practically the same, as determined by the period of hospital stay and the condition of the patient on discharge.

The incidence of surgical interference in the entire series was low (14 per cent) and was progressively lower in the younger age groups.

The percentage of symptomatic and anatomic cures was greater in the patients who were operated on than in those who were not.


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## MEDICAL PROGRESS

## OCCUPATIONAL DERMATOSES


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INDUSTRIAL medicine made remarkable progress as a result of World War I and World War II. Unprepared in 1917, it rapidly learned by trial and error, so that during the last few years of the later conflict, through preparation and research, it was able to maintain pace with the tremendous production of industry in wartime. Early recognition of the problems of health and hygiene contributed in no small part to the splendid industrial achievement in the United States. Prevention of industrial affections, by the control and elimination of hazards, enabled industry to perform almost a miracle, not only with less manpower but also with personnel less physically fit.

The skin received the first onslaught of industrial exposures. Dermatologists, especially those previously trained in industrial dermatology, were overwhelmed with work involving care of workers, inspection of plants and consultations with sanitary engineers and insurers. The results of their efforts at control and prevention, especially in cutting-oil hazards, rapidly became evident. The groundwork and teachings of the various committees for investigation of occupational dermatoses of the Council of Industrial Health of the American Medical Association early produced excellent results. The field work and publications of the Section of Dermatoses Investigation of the United States Public Health Service merit great commendation for its achievement. It is hoped that a vast amount of knowledge and statistics have naturally resulted from these efforts, and that the chemicals designed for human destruction may now be put to the advantage of the human race. Already, lethal gases and radioactive substances have been tried experimentally in the cure of fatal diseases such as leukemia and Hodgkin's disease.

Occupational dermatoses include injuries, inflammations, burns, stigmas and growths of the skin caused by occupational contact. Two committees, one appointed by the Section of Dermatology and


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Syphilology of the American Medical Association and the other formed by the American Dermatological Association, working in co-operation with the Council on Industrial Health, defined occupational dermatosis as "a pathological condition of the skin for which occupational exposure can be shown to be a major causal or contributory factor."[1] Recognition of the causative factor in an occupational dermatosis is important, since it has a direct bearing on the care of the case and on prevention of recurrence. It is estimated that 65 per cent of all occupational diseases are dermatoses.[2, 3]

Dermatitis in the form known as eczema is by far the most frequent of the occupational dermatoses. Dermatitis is an inflammation of the skin due to internal or external factors, the result of contact, ingestion, inhalation, injection, instillation or some other method of absorption.[4] The lesions, which are characterized by erythema, edema, papules and vesicles, may progress to simple scaling and desquamation or may become oozing and crusted and then desquamate; they may become secondarily infected with pustules or persist as infiltrated, excoriated, lichenified eruptions that prove resistant to treatment. Any one of the lesions, or any combination, may be present.

Occupational dermatoses are with few exceptions due to external agents. Dermatitis from such causes (dermatitis venenata) is so frequent that it requires classification. This classification considers first the type of agent, second the use of this agent and third the nature of the lesions produced. The second division is of little value because the agent may be employed under continually changing conditions; for example, paraphenylenediamine is extensively used in the manufacture of cosmetics, textiles and garments and in other branches of industry. The third division is also open to criticism because the same agent may produce varying degrees of disease depending on its strength as applied, the area to which it is applied and the method of application — that is, the degree of friction involved and the length of the period of exposure. An example is that of oils that cause folliculitis of the arms or cancer of the scrotum.

In general, the concentration of the irritant, the length of time it is applied and the condition under which exposure occurs serve to differentiate two types of dermatitis venenata — nonsensitization and sensitization. Just where the former ends and the latter begins is often impossible to determine. Nonsensitization dermatitis is caused by an irritant that affects practically all human skins. The irritant may be a mechanical or physical agent or a powerful chemical applied, either deliberately or accidentally, to the skin. The reaction is sudden and explosive. It is characterized by all degrees of inflammation and at times by the destruction of the cutaneous layers and even the subjacent tissues. The causative factor is almost always recognized and known to the patient; hence, recovery is fairly prompt, for with removal of the cause the eruption heals rapidly with little or no treatment. Eczema, variously known as sensitization dermatitis, contact dermatitis or contact eczema and allergic dermatitis or allergic eczema is an inflammation of the skin due to repeated exposures to substances that are normally innocuous. In this type of disease there is an initial exposure without resulting lesions, but subsequent exposures to even minimal quantities of the substance involved may result in reactions, manifested either at the local site of contact or as a generalized process. Every eczema is a dermatitis, but the converse is not true.

The actual causes of occupational dermatoses include mechanical, physical and chemical agents, flowering plants and their products and biologic agents. The chemical causes are divided into primary irritants and sensitizers.

A primary irritant is defined by the Council on Industrial Health[1] as follows: "When a substance in a given concentration, in a given vehicle and after a given manner and length of exposure produces clinically manifested irritation on the skin of the majority of persons not previously sensitized to that substance, then that substance is a primary irritant under the specified conditions." Primary irritants usually cause dermatitis after the first contact. They are the strong acids, alkalies, certain salts of heavy metals, solvents, essential oils, dye intermediates and some oils and greases.

A sensitizer is a substance that does not necessarily cause demonstrable cutaneous changes on the first contact but may effect such specific changes in the skin that, after five to seven days or more, further contact on the same or other parts of the body causes dermatitis. Sensitizers are chemicals that are innocuous to most skins. Sensitization may occur after a latent period of varying length — from days or weeks to months or even years.[5] The important sensitizers are resins, plastics, dyes, oils, soaps, accelerators, explosives, insecticides, plants, drugs and cosmetics. Experimentally no agent has been proved to be of value in desensitizing susceptible persons. It has, however, been the experience of one of us (J. G. D.) that in some industries the worker may become more or less resistant to outbreaks from contact with these substances and be able to continue his work, but that if he stops work and then returns to the same exposure, he may have an explosive recurrence of his eruption. Regarding this resistance there is apparent disagreement. Schwartz[6] has labeled this phenomenon "hardening," which he describes as the disappearance or the failure of reappearance of an allergic contact type of dermatitis in sensitized persons on repeated exposure in industry to the sensitizing chemical.

In plants manufacturing or using synthetic resin varnishes, there is a high incidence of sensitization dermatitis. Incompletely cured or incompletely polymerized resins and byproducts are responsible

for the eruption. Patch tests made with this material on workers suffering from dermatitis or who had an eruption due to contact with the synthetic resins elicited positive reactions. Other workers who were free from any skin eruption but who had previously had a sensitization dermatitis gave negative reactions.

Among workers with tetryl, 56 (11 per cent) of 500 employes in one plant developed dermatitis. In another 1905 (30 per cent) of 6364 workers developed dermatitis, and 1618 (85 per cent) of them returned to work with no further trouble. In a plant manufacturing TNT (trinitrotoluol), 10 per cent of the workers developed dermatitis, and all were able to return to work.[6] Probst and his associates,[5] on the other hand, found that tetryl workers did not develop tolerance. In aircraft workers suffering eruptions from dural, aluminum or zinc chromate primer, Hall[7] encountered no tolerance.

Hardening may be permanent but usually disappears if exposure is discontinued for any length of time. The degree of hardening varies. Exposures to higher concentrations of the sensitizing agent than that to which tolerance has been established may cause a recurrence of the dermatitis.

No attempt is made to present a complete review of all the causes of occupational dermatoses, since only the literature of the last few years is covered. Many causes are so well known and have been so completely eliminated from industry — for example, arsenic in the wallpaper industry — that the subject matter has been divided not according to occupation but according to the offending agent. A brief description of the occupations employing the particular irritant and of the cutaneous reactions to it follows.

## Mechanical and Physical Agents

### *Vibrating Tools*

Gurdjian and Walker[8] reported the occurrence of a circulatory disturbance of the hands of 6 women who used pneumatic hammers. After a few months to several years of exposure to the vibrations, the patients noted attacks of blanching and numbness of the fingers. In right-handed persons, the little, ring, middle and, sometimes, the index finger of the left hand and the tips of the fingers of the right hand were involved. The vibrating tool was held in the left hand, so that in left-handed persons the condition of the hands was reversed. The attacks of blanching often occurred during work, but usually came early in the morning or during washing, especially in cold weather. The condition is known by various names — dead fingers, white fingers, Raynaud's disease and traumatic vasospastic syndrome.

Hunter and Perry[9] observed white fingers in riveters, caulkers and fettlers using vibrating tools. Telford, McCann and MacCormack[10] reported "dead hands" in users of vibrating tools. Thirty-four cases developed after six to forty months' use of high-speed vibrating tools. These authors recommend that workers who have handled such tools for nine months stop this type of work.

The industries employing pneumatic hammers, high-speed vibrating tools (those operating at 2000 to 3000 r.p.m.), chisels, riveters, road drills and pounding and lasting machines are mining, quarrying, road-making, shipbuilding and shoemaking establishments, locomotive and other work shops and airplane factories. This condition is also said to occur in telephone operators,[8] but not in our observation of numerous operators.

### *Glass Fiber*

Champeix[11] reports mechanical irritation from glass fiber. Manifestations consisted of cutaneous erythema with pruritus, blepharitis and rhinopharyngeal irritation. In the weaving of glass fiber, the dust is not so much harmful as annoying because of the almost incessant itching.

In the manufacture of camouflage nets,[12] cutaneous reactions to spun glass (fiber glass or glass wool) appeared in a group of women handling it. The reactions observed were itching, without demonstrable lesions, and folliculitis. Spun glass is composed of fine threads that float in the air like dust and adhere readily to the skin.

### *Asbestos Materials*

Workers handling asbestos materials — especially amosite, a natural form of asbestos — develop painful warts or corns.[13] Of 167 workers, 99 presented one of more lesions. Those affected feel a small, splinterlike foreign body in the tips of the fingers, extraction of which is usually unsuccessful. In less than two weeks, a small cornlike tumor appears at the site of injury. This lesion grows slowly and is tender to pressure. When cornification is pronounced, small corns appear on the fingertips and knuckles.

Klauder and Hardy[14] report similar lesions in pipe coverers who handle asbestos with ungloved hands. Workers call this lesion an asbestos corn or wart. The lesion represents a foreign-body reaction to spicules of asbestos that penetrate the skin.

### *Chlorinated Hydrocarbons*

The eruption caused by the chlorinated waxes — chlorinated naphthalene, diphenyl and diphenyl oxide — is called chloracne or cable rash. Differentiation of this eruption and that seen in ordinary acne vulgaris is done by obtaining a history of exposure to the hydrocarbon in question and by finding acneform lesions in areas behind the ears and on the nape of the neck, the buttocks, thighs, knees, abdomen and lower back. These sites are not involved in acne vulgaris.

In a navy yard, over 200 cases of chloracne were observed.[15] The incidence of this rash has increased

because of the great acceleration of the shipbuilding program. Workers come in contact with waterproofed, fireproofed cable impregnated with chlorinated naphthalene, chlorinated diphenyl or chlorinated diphenyl oxide.

Folliculitis was caused by chlorinated hydrocarbons in 5 of a series of 1113 cases of cutaneous disease reported by Klauder[16] in 1943. In an additional 1184 cases reported by Klauder and Hardy[14] 3 cases were chloracne.

The lesions in chloracne are due to chloro compounds deposited as fine particles or condensations on the skin from fumes and solutions.[17] Such compounds plug the follicular orifices and by their keratogenous action cause the formation of comedones. Yellow cysts develop as a result of mechanical plugging of the openings of the glands and the actual keratinization of their walls. Preventive measures in general are the proper selection of workers, cleanliness, the wearing of clean clothing and adequate ventilation. Workers with a tendency to seborrhea or acne should be excluded from this type of work, since they are as a rule more susceptible than others to acneform eruptions.

*Cutting Oils*

The cutting oils are the most frequent cause of dermatoses in factories engaged in the making and machining of metal parts. In the manufacture of the actual materials for airplanes, tanks, guns and vehicles this vexing problem confronts the industrial physician.

There are two large groups of cutting oils, the soluble and the insoluble. The former consists of a mixture of sulfonated mineral oil and soap containing resin, sulfonic acids and preservatives — phenol, creosol or nitrobenzene — when animal or vegetable fats are used in the oils. These oils are mixed with water and allowed to flow continuously over the material under operation. Their chief function is the cooling of the metal parts. The insoluble oils are mixtures of mineral, animal and vegetable oils, sulfur and chlorine. The last two chemicals enable deeper cuts to be made into the metal without harming the cutting tools. In the oils containing animal or vegetable oils, inhibitors such as phenolic amines are added to prevent rancidity.

Mineral oils cause defatting of the skin. The small amount of animal or vegetable oils present tends to counteract this effect. The cutting oils produce lesions of the skin owing to mechanical interference with or plugging of the pores. Dermatitis, both sensitization and nonsensitization, irritations, abrasions due to metal slivers in the oils and secondary infections are caused by cutting oils. For the sake of simplicity the skin manifestations of these oils are mentioned under this heading.

In a group of 450 cases of occupational dermatosis reported as to cause, 45 (8.5 per cent) were due to petroleum products.[16] Thirty-six cases were of the oil folliculitis type. There were 1 case each of generalized melanosis, localized melanosis, keratosis and epithelioma and 7 cases of nonsensitization dermatitis from cutting oils. Oil folliculitis was the most frequent form of dermatosis in workers exposed to lubricating oils and greases and was severe in most cases.

Klauder[16] examined 128 men operating automatic screw machines and found comedones, small follicular, horny papules, acnelike papules, pustules, pyoderma and scars resulting from destruction of the follicular orifices. In only 19 per cent of the workers were no lesions found. In another study of 50 cases of folliculoses,[14] 42 were found to be caused by mineral oil in machine operators and men engaged in quenching metals. Two of these cases were complicated by cellulitis and lymphangitis.

Peck[17] reports that dermatitis from cutting oils, especially those of the insoluble type, occurs more frequently than any other occupational skin disease. The lesions oftenest seen are folliculitis and comedones on the hairy portions of the arms and the anterior surfaces of the thighs.

Schwartz,[18, 19] after making a survey of the various skin hazards in war industries, reports that cutting oils are frequent causes of dermatitis. They defat and irritate the skin and plug the pores. The sulfur, chlorine or rancid animal or vegetable fats in the oils are irritating. Phenols, creosols, nitrobenzene and other inhibitors present in the oils may sensitize the skin, and steel slivers in dirty oils often cut the skin.

Allergic eczemas are the least frequent types of cutting-oil dermatitis.

Some mineral oils have keratogenic properties and cause papillomas and warts. Schwartz[18] found these lesions in 10 per cent of the workers, an exceedingly high percentage that does not correspond with the observation of others. Certain oils are carcinogenic, but fortunately the mineral oils of North America are low in the carcinogenic scale. Some cases of skin cancer, however, occur in the United States as the result of contact with mineral oil. There was 1 case of epithelioma in 45 cases of dermatosis due to petroleum products.[16]

Prevention consists essentially in reducing to a minimum the contact between workers and irritants. Machines should be kept free from grease and dirt by daily cleaning. Oil should not be allowed to accumulate on the floor. Guards should be attached to machines to prevent oil from splashing on workers. Adequate washing facilities and clean work clothes should be provided, and impervious sleeves and aprons should be worn. Klauder[16] advises the use of two protective ointments, the first being

smeared thickly over the entire forearm as a base for the second. These ointments are composed as follows:

| FIRST OINTMENT | gm. | SECOND OINTMENT | gm. or cc. |
|---|---|---|---|
| Zinc oxide | 25 | Ethyl cellulose | 5 |
| White petrolatum | 25 | Mastic | 8 |
| Kaolin | 50 | Castor oil | 1 |
| | | Acetone (technical) | 86 |

The second solution is applied with a small brush and dries, leaving a film that is repellent to both water and oil.

*Abrasions, Cuts, Friction and Pressure*

Abrasions, lacerations and fissures often occur throughout the fish industry, and subsequent infections are frequent and disabling.[20] Metal slivers in the cutting oils produce cuts of the skin.[18]

Stigmas peculiar to certain occupations are due to pressure, friction, repeated injuries and joint deformities. Ronchese[21] enumerates the following trades with their stigmas. Stone chiselers have a characteristic large, soft, rubbery callus at the proximal end of the left little finger. Painters and paperhangers have an elongated callus in the center of each shin, owing to pressure of the rung of the ladder. Jewelers, engravers, ringmakers and stone setters show a characteristic large, heavy callus in the center of the right palm, produced by their most important tool, the plier. Mechanics, machinists and toolmakers as a rule have rough hands showing cuts and abrasions. The nails are worn down, the fingertips are often raw, and the palms are studded with metal slivers from handling and filing metal parts. Foundry workers and melters of metals show a vivid-red discoloration (erythema ab igne) of the forearm, which is continuously exposed to intense dry heat in the act of pulling metals from ovens. The shins of junk collectors, garbage men, truck drivers and railroad conductors show varying degrees of abrasions, cuts, scars and pigmentation. This condition is due to frequent trauma to the shins.

Perhaps the most important physical agent of this era from the point of view of health is radioactive material. Its effect on human tissue has merited and will continue to merit careful consideration. In a nonindustrial paper Dunlap[22] presents an excellent review of the injuries resulting from roentgen rays and radioactive substances. Years ago industrial physicians learned the deleterious effects of the alpha and beta rays emanating from radium on the health of persons who swallowed or inhaled radioactive substances or took them into their bodies in other ways. Physicists have succeeded in rendering practically all the known elements temporarily radioactive by exposing them on a percursor to the enormous energies generated by the cyclotron. Thus a new industrial hazard is incurred by persons working with or near cyclotrons or their radioactive products.

The character of tissue damage appears to be identical with the injuries resulting from exposure to roentgen rays or radium. In the manufacture of roentgen-ray tubes there is an obvious hazard to employees for which in most factories there is adequate protection. Detection of flaws in metal castings and welds by roentgenograms and radium photography requires the use of enormous doses of radiation, from which serious effects result if there is no intelligent medical supervision. Insurers in Boston learned this several years ago when an efficiency expert introduced an x-ray machine to speed up a certain process, with resulting radiodermatoses to workers exposed. The manufacturers of luminous dials have learned the terrible toll among their workers from inexperience in handling minute amounts of radioactive material. The manufacture and handling of radium applicators and needles involve exposure not only to radiations but also to radon gas in the atmosphere and to the chance inhalation or ingestion of particles of radium. The mining and handling of radioactive ores should be closely supervised. Although no medical difficulties have been encountered among the miners of pitchblende, the chief source of radium, workers in the cobalt mines of Germany suffered for centuries from an obscure disease of the lungs, recently identified as cancer, that was probably due to inhaled radon gas. Chemists who have worked with radium and related compounds have suffered from burns and anemias. Human curiosity and careless supervision of roentgen-ray equipment, especially the fluoroscope, may be disastrous. A number of workmen in a large industrial plant on the West Coast were seriously burned in a recent series of laymen's experiments conducted with a fluoroscopic machine in the first-aid room.[22]

## Flowering Plants and Their Products

*Woods and Weeds*

Piorkowski,[23] observing that dermatitis is frequent among workers handling tropical woods, reported 5 cases in Indians handling mvule wood, which is similar to teak and oak in its uses. The clinical picture was that of an acute dermatitis venenata covering the exposed parts of the face, neck, forearms and hands and causing severe itching. The eyelids were swollen. In 1 case catarrhal conjunctivitis and rhinitis were present. In 4 cases the onset was sudden and the attack lasted for one or two weeks; in another it lasted for six months. The eruption developed subsequently and consisted of chronic eczematous changes, marked pruritus and lichenification. The chemical agents responsible are the alkaloids or nonsaturated resinous acids.

Howell[24] in a recent article on contact dermatitis reports that in the Southwest an eczematous contact dermatitis from sensitization to vegetation is a

frequent occurrence. The condition is an occupational hazard among ranchers, farmers and workers in the oil fields. Howell reported 35 patients who were sensitive to weeds — some to several and others to only one. This was brought out by patch tests with the oleoresins of forty-five different weeds.

*Extracts and Resins*

*Cashew-nut-shell oil.* The oil of the cashew nut shell is obtained either by roasting the shells, the general practice in India, or by solvent extraction, which is said to produce a much higher yield of oil. The oil is a mixture of 90 per cent monohydric phenol (anacardic acid) and 10 per cent polyhydric phenol (cardol). The cardol fraction is believed to contain the major irritant properties of this oil and is related chemically to urushiol, the irritant principle of poison ivy. Cashew-nut-shell oil has been proved to be a primary irritant and a sensitizer.[25] Two patients had to be hospitalized as a result of patch testing with this substance. The oil would be invaluable in industry except for these deleterious properties.

An investigation[26] of the manufacture and use of this liquid was conducted when occupational dermatitis due to resinous varnish made from cashew-nut-shell oil and formaldehyde occurred among many workers handling electric components insulated with this varnish. One plant visited had been using cashew-nut-shell oil for nineteen years, manufacturing a variety of resins including insulating varnish, dusts for brake linings, molding powders and millable rubbery compounds. These materials represent products of cashew-nut-shell oil and other chemicals in various stages of polymerization and condensation or cure. In this plant 4 of every 10 new workers handling the raw oil develop dermatitis, and one quarter of the attacks are severe. Patch tests with this oil on 15 subjects showed marked reactions at the end of twenty-four hours in all subjects, which proves that it is a primary skin irritant. Cured varnish caused a positive patch-test reaction in only 1 case.

Lockey[27] reports contact dermatitis as developing in 9 men who for some time had been using so-called "H. varnish E. 107," which contains cashew-nut-shell liquid as one of its main bases. Patch tests revealed that all these men were sensitive to the oil; 7 reacted to the varnish from the barrel that was being used at the time the dermatitis appeared, whereas none reacted to varnish used previously. It is probable that the oil had not been properly broken down in the manufacturing process.

*Resins.* Over twenty-six hundred objects used in this country are made of resins.[28] The phenol formaldehyde and urea formaldehyde resins account for many cases of contact dermatitis during the process of their manufacture. Dermatitis appeared in 78 of 150 workers engaged in making tubes for launching rocket bombs from airplanes; these tubes were made from paper glued together with a low-polymerized phenol formaldehyde resin.[29]

Cranch[30] states that the outstanding source of trouble in the use of plastics is local — that is, dermatitis. The finished products made from plastics are seldom irritants, and in only a few cases do they cause sensitization with resulting dermatitis.

Hall,[7] in analyzing occupational dermatitis among aircraft workers, found that those using zinc primer reacted to zinc chromate or to the resins in the primer (phenol formaldehyde, a natural resin and a phtholic anhydride) or to both. Of those who reacted to the primer, 13 per cent were sensitive to one or more of the resins but not to the zinc chromate, and 15 per cent were sensitive to one or more of the resins and also to zinc chromate.

The dermatitis due to resins was uniform in its characteristics. The eyelids were affected in all cases, and the side of the upper two thirds of the neck and the cubital fossas in the great majority. The hand and fingers were affected in only 1 case, and the wrists and forearms in only 6. The average duration of exposure before onset of symptoms was seven months — less than that for zinc chromate. In 3 cases, however, the average duration was less than a week.

*Lemon-grass oil.* Mendelsohn[31] reports dermatitis in 8 machinists, carpenters and riggers who worked for three weeks on a ship recently arrived from India. Part of the cargo comprised tanks of lemon-grass oil, some of which was spilled. The cutaneous lesions appeared six to eighteen days after this work. The eruption consisted of discrete and confluent erythematosquamous patches of various sizes, some slightly vesicular. The lesions were on the face, on the flexor aspects of the lower two thirds of the forearms and about the ankles. Three men had edema of the eyelids, and 1 also had considerable dermatitis of the penis, scrotum and upper part of the thighs. Two men reported that members of their families had similar rashes, evidently following contact with their contaminated work clothes. Four workers were patch tested with pieces of pine wood soaked in lemon-grass oil, and all showed strongly positive reactions. Lemon-grass oil has many uses; it is employed in the manufacture of perfume and iomene and as an adulterant for lemon oil.

*Fruits and vegetables.* In the picking, packing and canning of fruits, various forms of cutaneous irritation are encountered.[32] In the citrus fruit industry, dermatitis is caused by the irritant oil in the rind of lemons, oranges, limes and grapefruit. Among the pickers of such fruits, dermatitis is produced by the thorns on citrus trees and by the insecticides and fumicides sprayed on them. Paronychia occurs among canners of these fruits. Pineapple peelers develop dermatitis from the sharp spears on the fruit, and the high sugar content of

pineapples also causes dermatitis. The sharp fuzz on peaches is irritating to pickers and canners, and the sugar in the juice is also irritating to the skin.

Dierker[33] reports the results of an investigation of dermatitis occurring in a small plant producing marmalades and jellies. No skin irritation was encountered among the workers until a load of Seville oranges was converted into marmalade. The oranges were washed and dried, the skins were removed, and the fruit was cut up. In this operation 5 of 10 workers developed diffuse erythema of the dorsum of the hands and wrists and the webs of the fingers. Patch tests on 8 normal subjects with extracts of the orange skins as well as juice from the fruit elicited no reaction. The workers with dermatitis were not tested. The patch tests, however, gave evidence that no primary irritant was the cause of the dermatitis. It was thought that the high acidity of this species of orange, together with the continuous wetting of the hands, produced the dermatitis. The use of a protective cream against acid, dipping the hands in a weak solution of sodium bicarbonate and the wearing of gloves prevented further trouble.

Peck[34] reported dermatitis arising from the dehydration of potatoes. He inspected six plants engaged in this process, only one of which used lye in the peeling process. The potatoes were washed, precooked, immersed in a 10 per cent lye solution and peeled by friction in a rotating drum. The final washing removed all traces of lye. In the other five plants the potatoes were precooked and peeled by rough rubber rolls, no lye being used. After peeling the potatoes were brought to the trimming tables by moving belts, where workers trimmed off the remnants of the peel with special knives; their hands were constantly wet with water and potato juice in this operation. Dermatitis of the hands, which occurred in 24 women workers, started in the hand holding the potato in the trimming operation; it consisted of redness, scaling and sometimes vesicles in the web spaces, resembling erosio interdigitalis blastomycetica as the condition progressed. Patch tests with slices of potato, cooked and uncooked, on 12 workers and 5 controls were negative.

This dermatitis is due to long exposure to water and potato juice, which results in maceration of the skin. Workers whose hands are thus constantly wet eventually develop dermatitis and show interdigital maceration and redness suggestive of erosio interdigitalis blastomycetica. Patch tests are unnecessary, for no such test can reproduce the occupational exposure. The workers affected include dishwashers, salad makers, vegetable salesmen, cooks, peelers and canners of vegetables and young housewives — by far the largest group encountered in the private practice of one of us (J.G.D.).

Dermatitis occurred in 17 women who worked with carrots.[35] The cutaneous eruption appeared on the palms, the thenar and hypothenar eminences, the dorsum of the hands and forearms and in some cases even the neck, face and eyelids. The incubation period was generally from seven to ten days but sometimes lasted for several months. When workers were removed from contact with either cooked or uncooked carrots, the dermatitis cleared up. Patch tests with carrots, cooked and uncooked, indicated that they were definitely the cause of the eruption.

Thirteen women handling partially decayed parsnips for three or four weeks developed dermatitis.[36] When the parsnips were cut, the juice ran down the back of the left hand and sometimes down the knife to the right hand. On exposure to the sun, redness, swelling and then pigmentation developed at these sites. Six other women similarly exposed were not affected.

*(To be concluded)*

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# MASSACHUSETTS MEDICAL SOCIETY

## PROCEEDINGS OF THE ONE HUNDRED AND SIXTY-FIFTH ANNIVERSARY

### May 21, 22, and 23, 1946

THE one hundred and sixty-fifth anniversary of the Massachusetts Medical Society was observed in Boston on Tuesday, Wednesday and Thursday, May 21, 22 and 23, 1946, at the Hotel Statler.

Seventeen hundred and fifty physicians were registered.

### Tuesday, May 21

The first scientific session began at 2:00 p.m. At 5:00 p.m. the supervising censors met in Parlor D.

The Cotting Supper was served to 185 councilors in Parlors A and B. The annual meeting of the Council was held in the Georgian Room at 7:00 p.m. There were 191 councilors in attendance.

### Wednesday, May 22

The second scientific session was held under the joint co-chairmanship of Dr. W. Jason Mixter and Dr. C. J. E. Kickham. The attendance varied from 500 to 600.

The one hundred and sixty-fifth annual meeting of the Society was held at 11:00 a.m. in the Georgian Room. Dr. Reginald Fitz presided. The attendance was 500. Dr. Fitz spoke at length on "The State of the Society." The Annual Oration, entitled "Gastric Surgery," was delivered by Dr. Frank H. Lahey. The annual luncheon was served in Parlors A, B and C to 219 fellows.

The third scientific session was held in the Georgian Room from 2:00 to 4:00 p.m. under the co-chairmanship of Dr. Elmer S. Bagnall and Dr. John Fallon. The attendance was 600. The Shattuck Lecture was delivered at 4:00 p.m. by Dr. John B. Youmans, whose subject was "Nutrition and the War." (This lecture appeared in the June 13 issue of the *Journal.*)

The annual dinner was held in the Georgian Room at 7:00 p.m. with 525 in attendance. The principal speakers were Dr. Roger I. Lee, president of the American Medical Association, and Dr. Elmer S. Bagnall, former president of the Massachusetts Medical Society.

### Thursday, May 23

A general scientific session began at 9:00 a.m. and continued until noon under the co-chairmanship of Dr. Archibald McK. Fraser and Dr. Arthur J. Gorman. The attendance was between 550 and 600.

From noon until 2:00 p.m. a series of section meetings and luncheons was held. The Section of Medicine, under the chairmanship of Dr. Albert A. Hornor, met in Parlor B. Fifty-one sat down to luncheon. The Section of Surgery, under the chairmanship of Dr. Charles F. Twomey, met in the Salle Moderne. The attendance was 133. The Section of Pediatrics, under the chairmanship of Dr. Floyd R. Smith, met at the Junior League, Zero Marlborough Street. The attendance was 55. The Section of Obstetrics and Gynecology met in Parlor A, under the chairmanship of Dr. Arthur F. G. Edgelow. The attendance was 52. The Section of Radiology, under the chairmanship of Dr. George Levene, met in Room 400. The attendance was 28. The Section of Physical Medicine, under the chairmanship of Dr. Arthur L. Watkins, met in Parlors D and E. The attendance was 12. The Section of Dermatology, under the chairmanship of Dr. Bernard Appel, met in the Hancock Room. The attendance was 38. The Section of Anesthesiology, under the chairmanship of Dr. Sidney C. Wiggin, met in Parlor C. The attendance was 29.

From 2:00 to 5:00 p.m. a general scientific session was held in the Georgian Room under the co-chairmanship of Dr. Frank R. Ober and Dr. Harry Blotner. The attendance was 600.

There were seventeen scientific exhibits and eighty-four technical exhibits.

The motion pictures, as usual, attracted a large number.

The special list of officers, standing and special committees, councilors, censors, admissions and deaths are appended.

Michael A. Tighe, *Secretary*

## Annual Meeting of the Society

The one hundred and sixty-fifth annual meeting of the Massachusetts Medical Society was called to order by the president, Dr. Reginald Fitz, in the Georgian Room of the Hotel Statler, Boston, at 11:00 a.m., May 22, 1946. There were approximately 500 Fellows present.

The Secretary submitted the record of the last annual meeting held on May 23, 1944, as published in the *New England Journal of Medicine*, issue of August 3, 1944. It was moved and seconded that the record be adopted. It was so ordered by vote of the fellows.

The Secretary reported that the membership of the Massachusetts Medical Society, as of May 23, 1945, was 5786 and as of May 22, 1946, 5989. He said that 85 fellows had died, 16 had resigned and 6 had been deprived of membership. He added that 302 new fellows had been admitted and that 8 had been reinstated. It was regularly moved and seconded that the Secretary's report be accepted.

It was so ordered by vote of the fellows.

At the request of the President, the Vice-President, Dr. W. Jason Mixter, assumed the chair. Dr. Mixter spoke as follows:

> Members of the Society, I should like to take a short moment to make a personal report on the activities of your president during the past year. You will find in his report that the condition of the Society is healthy. I am sure that one of the major reasons for this condition is the fact that Dr. Fitz has given lavishly of his time and energy to the business of the Society. He has used tact where tact was needed. He has used the authority of his office for the benefit of the Society where it was needed, and I feel that the Society owes a very large vote of thanks to Dr. Fitz for his activities as our president. Mr. President, I ask you to give your report on "The State of the Society."

Dr. Fitz responded as follows:

> For the third time in the last hundred years the Society, on the occasion of its annual meeting, is in a period of readjustment following a great war.
>
> In 1865, the finish of the War between the States was so close that the fellows scarcely as yet realized the full impact of peace. This meeting was poorly attended — in part because of the disturbed state of the Nation and in part because the American Medical Association was to assemble in Boston during the following week. There was no annual dinner, nor did the scientific program lay any great emphasis on war medicine. It is interesting, however, that the most debated topic under discussion by the American Medical Association dealt with specialists: what a specialist was, and what he was supposed to do.
>
> At the annual meeting in 1919, military medicine received much more consideration. Nearly a third of the doctors in Massachusetts had experienced active military service; they seemed eager to resume their civilian occupations as promptly as they could but were equally willing to hear what the war had added to medical knowledge. The *Proceedings of the Council*, on the other hand, do not suggest that any very vital problems had developed, and indeed, except for one instance, the entire meeting was largely devoid of extraordinary contributions. It was at the annual dinner that Dr. Samuel B. Woodward, who was president, presented ideas not often mentioned. These ripened slowly with time and still promise important results. He said that all fellows should take a more active interest than heretofore in legislative matters and should have no reluctance in stating their opinions at the State House or anywhere else on matters concerning the health and welfare of their fellow citizens.
>
> The past year has been an eventful one. As in 1919, approximately a third of our physicians have recently experienced active military service, and as in 1865, the matter of specialism is of vital interest.
>
> A preview, obtained by questionnaires from the American Medical Association, suggested that a new type of practice might presently become manifest. The men in service wrote that they hoped, on discharge, for an urban type of practice rather than for a general practice in rural districts and that they looked forward to opportunities for postgraduate education with the idea of becoming specialists or of working in well organized groups. The Society realized these trends and, through the Postwar Planning Committee, arranged for appropriate steps to assist in their development, should these be necessary.
>
> Our work in postgraduate education is an important milestone. By using available facilities all over the Commonwealth, we have administered, successfully, a variety of postgraduate courses, which have proved both popular and refreshing. More than a thousand doctors have had opportunity to bring themselves up to date on current medical ideas through expertly delivered lectures. In the words of the first Dr. John Warren, the result is that the Society has gone farther than ever before, for the benefit of its fellows, "to promote medical and surgical knowledge by encouraging a free intercourse with the Gentlemen of the Faculty."
>
> The Society has watched the return of its medical officers with care as well as with pride. Apparently the majority of our veterans are as eager as were their older brothers to resume civilian work where they left it and as promptly as they could; up to the present time, they have made no significant attempt either to overspecialize or to overexpand group practices. To be sure, many young veterans are hoping to complete the program of training on which they had embarked before they entered military service. For these men, especially, the Bureau of Clinical Information fills a useful function. Opportunities for the supervised, long-continued type of hospital training demanded by specialty boards are, by necessity, limited. The Bureau of Clinical Information has kept track of such opportunities as there are, and the Postwar Planning Committee has endeavored to create new ones locally. A number of veterans, however, have been disappointed by their inability to find at once exactly the residencies that they hoped would be available. All that can be said is that the machinery of the Society is working to help such men as best it can.
>
> The Postwar Loan Fund represents another attempt by the Society to assist returning veterans. The problems that face a man who has been out of civilian circulation for a few years are bewildering, not only in matters of education but also in the more earthy ones of finding a house in which to live or an office in which to practice or equipment with which to work. For these latter details, the Postwar Loan Fund is of assistance: at least it may provide a much-needed sense of financial security.
>
> Our executive work has continued to go forward through usual channels. As Dr. Lee pointed out two years ago, the Society operates through committees. During the past year, twenty-seven committees have functioned industriously on our behalf. It is the President's privilege to be present at all committee meetings that he desires to attend, and thereby he is given unequaled opportunity to keep abreast of the Society's activities.
>
> Most of these committees have functioned with zeal far beyond the ordinary call of duty. I have already mentioned the educational accomplishments of the Postwar Planning Committee. This committee, through a subcommittee established to work in co-operation with subcommittees from the committees on Legislation and Public Relations, also struggled with a hydra that made its appearance at the end of World War I.
>
> At the annual meeting in 1919, there was talk of the physical imperfections in the youth of America that had become manifest through examinations for the draft. This general subject gradually attained national popularity through one or another of its many heads until finally President Truman's National Health Program of 1946 emerged. The opinion of the Society on a matter so important to the nation was crystallized by these subcommittees, by their parent committees and finally by the Council, in the form of a short pamphlet. Herein the Massachusetts Medical

Society offers certain basic principles to govern proper medical-care plans and expresses its views concerning national legislation now pending. This is an important document, interesting, chiefly, because it sets forth basic principles concerning the care of sick people to which the majority of fellows wish to subscribe, and interesting, also, because it shows so clearly the Society's philosophy. We wish everyone to have every known essential preventive, diagnostic and curative medical service of high quality. We believe that this can be best attained by a gradual process of evolution rather than by sudden revolution. We believe that it is the responsibility of persons who are able to do so to procure for themselves many phases of medical care by direct payment or by voluntary insurance against their costs. And finally, we continue to cherish an old ideal: to receive the best kind of medical care we are convinced that every patient must have the right to select the physician or hospital to which he chooses to entrust the care of his health.

The activities of the Committee on Legislation are perennial. The Society has come a long way since Dr. Woodward reminded us of our obligations as citizens. Fellows are less diffident than they used to be at hearings or indeed at meeting legislators as men to men. Several district societies have entertained their representatives or senators at dinner — an important method of breaking down existing barriers; members of the Great and General Court prove agreeable dinner companions, appearing fully as eager as we are to improve the health and welfare of our citizens and willing to hear our views.

The Committee on Legislation has experimented with a new method of organization. During the past year it has functioned through an executive committee and through subcommittees. In this manner each member of the committee has been kept informed of legislative matters in general, and each subcommittee has worked intensively on such bills as were assigned it. One subcommittee, for example, studied, with particular care, antivivisection bills, another, bills concerned with hospital staff appointments, and a third, the chiropractic bill. The Executive Committee has integrated the work of its subcommittees with the work of the parent committee, drawing heavily on Mr. Charles Dunn for legal and technical advice. This method of organization seems reasonable, promises to yield good results and warrants further exploration.

Several months ago, Major General Paul R. Hawley requested the Society to appoint a committee to confer with his office about the medical care of veterans. The Council authorized the appointment of such a committee, which in turn worked most industriously. As a result it is hoped that certain veterans may be free, in case of need, to employ civilian physicians and hospitals of their own choice in Massachusetts. Their hospital expenses will be met through the Blue Cross as agent, and their professional bills will be met through the Blue Shield as agent, according to a fee schedule established to cover almost all professional services that can be offered. The construction of so comprehensive a fee table proved a difficult, time-consuming task, requiring the welding together of many different opinions from various groups of physicians; in its final form it represents, to a high degree, conscientious and painstaking effort.

At its stated meeting in October, the Council voted to blaze a new trail. The committees on Legislation, Postwar planning and Public Relations submitted a joint resolution advocating the creation of a new position in the Society: that of director of medical information and education. The need for a full-time worker in our executive offices had become increasingly apparent for several years, although the definition of the exact duties of such a person was more difficult. The Council supported the resolution offered by these committees and voted to appoint a special committee to nominate a suitable candidate for the new post. This committee as yet has made no nomination, preferring to act slowly on a matter of such importance; the establishment of this position is certain to have far-reaching results.

At the stated meeting in February, the Council made another departure from old ways. It advocated a plan whereby certain licensed physicians in Massachusetts who were graduates of unrecognized medical schools might obtain hospital privileges. This action represents a broad-minded attitude in the treatment of an old sore point. Everyone knows that Massachusetts, in the past, has licensed a number of physicians who graduated from unrecognized medical schools, and that by last year's legislative action their future source of supply was jugulated. On the other hand, such doctors, already licensed, will continue to practice and are the family physicians of many patients. Dr. H. Quimby Gallupe, of Middlesex South, to aid these men and their patients, presented an ingenious plan by which courtesy-staff hospital privileges under carefully supervised regulations could be made more generally available to them. The Council adopted the plan, and the Committee to Meet with the Massachusetts Hospital Association has been assigned the duty of furthering it. Already a series of meetings with hospital representatives has commenced at which the details of the plan will be discussed around a small table with the informality that is possible when small groups are involved. This is an important step. If both hospitals and fellows co-operate to activate this plan, the Society will be able to take credit for having promoted a liberal and unselfish program primarily designed for public benefit but also having substantial educational value.

Twenty-five years ago, the *New England Journal of Medicine* became owned by the Society and subsequently has been published by it. We have been wise in our choice of editors. Dr. Walter P. Bowers laid the foundation for the *Journal's* present success, and Dr. Robert N. Nye has continued to carry forward his aims. During 1945, about 18,000 readers received the *Journal* each week, and the enthusiasm with which our published articles and editorials have been received all over the world is indeed flattering.

I have selected these few features in the Society's most recent history to emphasize how, little by little, our influence and interests expand. In spite of so much exertion our physical condition is satisfactory. Our membership increases steadily, our finances are in good order, and our spirit is that of ardent youth tempered by our recorded age.

Each president, as he discusses the state of the Society at the annual meeting, by necessity renders his report with mixed feelings of pride and humility. For all his days he must remain proud that his colleagues have paid him the compliment of allowing him to occupy the highest office in the Society for a year. On the other hand, he is humble because he realizes how small a part he has played, even for those few months, in carrying on the efforts of so large an organization as the Society has grown to be.

The district societies, the Council, the committees with their devoted chairmen, Robert N. Nye, of the *Journal*, Robert St. B. Boyd, the executive secretary, Miss Cowles, tireless in keeping our records straight, Eliot Hubbard, Jr., ever watchful of our purse and, above all, Michael Tighe, tolerant and wise, are our flag-bearers. They are the ones who keep our ideals virile and continue to make Dr. Jacob Bigelow's words of a century ago so timely: "I give you, gentlemen, the Massachusetts Medical Society; old but not infirm, numerous but not divided. May it long continue to be what hitherto it always has been: the protection and pride of those who are worthy to be its members."

The President resumed the Chair. At the direction of the President, the Secretary read the following amendment to the by-laws:

> The by-laws of the Massachusetts Medical Society are hereby amended by adding a new chapter, to be known as Chapter X. The chapter shall read as follows:
>
> Any provision or provisions of these by-laws may be temporarily waived, provided such waiver is not in conflict with the laws of the Commonwealth, provided the provision or provisions sought to be waived are clearly designated, provided the period during which such waived provision or provisions are to continue be definitely stated, provided notice of the proposed waiver accompany the call of the annual meeting and provided the members present at such an annual meeting unanimously consent.

The President explained that this amendment was legally before the Society for action. It had been

approved by the Council and had accompanied the notice of the annual meeting. Its intent, he continued, was to make the by-laws more flexible. The Secretary moved the adoption of the amendment. This motion was seconded and was so ordered by vote of the fellows.

Dr. Thomas A. Foster, delegate from the Maine Medical Association, was introduced. He responded as follows:

> Mr. President and members of the Massachusetts Medical Society: the Maine Medical Association and the Massachusetts Medical Society have had a long and friendly relation, the Maine Medical Association being born in 1820, when Maine became separated from the Commonwealth of Massachusetts. During that time we have sent many delegates to the annual meetings of the Massachusetts Medical Society.
>
> With the permission of your president I should like to read some extracts from the report of a delegate to a meeting of the Massachusetts Medical Society, Thomas Albert Foster, of Portland, in 1871.
>
> At twelve noon a meeting was holden in Lowell Institute at which several essays were read by young men who, from the character of their papers, I judge, will make their mark in the profession of medicine in that city. One of them, Dr. Fitz I think they called him, I was told had recently returned from Germany where he had been spending some time under the instruction of Professor Burkhardt. His essay was on tuberculosis and was an able declaration.
>
> The delegate speaks at length of other parts of the meeting and made a conclusion that I am not altogether sure is courteous.
>
> As a whole, I think the Massachusetts Medical Society, although having among its members many of the ablest physicians and surgeons of the Country, is very far behind our own association in point of work. As good as the papers were, the subjects upon which they were written were by no means exhausted by their authors and still there was no discussion of any interest upon any of them. I am sure I can return to the Maine Medical Association and find they still have the ablest physicians and surgeons and their papers are thoroughly prepared and exhausted. The discussion was enlightening.

The President read the list of officers elected by the Council on May 21, 1946.

Dr. Fitz then introduced the incoming president, Dr. Dwight O'Hara. Dr. O'Hara responded as follows:

> President Fitz and fellows of the Society: I told the Council last night that my main endeavor will be to come as nearly as I can to following in the performance and pattern of the administration that Dr. Fitz has set up during the past year. I know that I shall not attain his efficiency, but I shall endeavor to do so and I shall count on your support therein. Thank you.

Dr. Fitz then introduced Dr. Edward P. Bagg, the president-elect. Dr. Bagg responded as follows:

> Mr. President and fellows: this in indeed a historic occasion. I believe that I am the first pediatrician who has been put in line for the presidency. I shall try to live up to all the good beginnings that pediatricians are trained in and let the chips fall where they may. I hope that I shall not disappoint the electors.

At this point in the meeting the President introduced Dr. Frank H. Lahey, who presented the Annual Oration. His subject was "Gastric Surgery." The address was brilliantly given. It held the close attention of the audience through its entire delivery. (The oration appeared in the June 20 issue of the *Journal*.)

Dr. Fitz declared the one hundred and sixty-fifth annual meeting of the Massachusetts Medical Society adjourned at 1:00 p.m.

MICHAEL A. TIGHE, *Secretary*

# APPENDIX

## OFFICERS FOR 1946–1947

PRESIDENT: Dwight O'Hara, Waltham. Office, Boston (15), 416 Huntington Avenue.
PRESIDENT-ELECT: Edward P. Bagg, Holyoke, 207 Elm Street.
VICE-PRESIDENT: Isaac S. F. Dodd, Pittsfield, 34 Fenn Street.
SECRETARY: Michael A. Tighe, Lowell. Office, Boston (15), 8 Fenway.
TREASURER: Eliot Hubbard, Jr., Cambridge, 29 Highland Street.
ASSISTANT TREASURER: Norman A. Welch, West Roxbury. Office, Boston (15), 520 Commonwealth Avenue.
ORATOR: Leland S. McKittrick, Boston (16), 205 Beacon Street.

## COMMITTEES ELECTED BY THE DISTRICTS

**Executive Committee of the Council** — Established 1941 (members *ex-officiis* and one councilor and alternate elected by the councilors of each district medical society).
PRESIDENT: Dwight O'Hara, Waltham. Office, Boston (15), 416 Huntington Avenue.
PRESIDENT-ELECT: Edward P. Bagg, Holyoke, 207 Elm Street.
VICE-PRESIDENT: Isaac S. F. Dodd, Pittsfield, 34 Fenn Street.
SECRETARY: Michael A. Tighe, Lowell. Office, Boston (15), 8 Fenway.
TREASURER: Eliot Hubbard, Jr., Cambridge, 29 Highland Street.

*Term Expires 1947*

BERKSHIRE: Isaac S. F. Dodd, Pittsfield, 34 Fenn Street. (Alternate: Clement F. Kernan, Pittsfield, 184 North Street.)
FRANKLIN: John E. Moran, Greenfield, 31 Federal Street. (Alternate: Frank A. Millett, Greenfield, 40 High Street.)
HAMPDEN: William A. R. Chapin, Springfield, 121 Chestnut Street. (Alternate: Archibald J. Douglas, Westfield, 30 Court Street.)
MIDDLESEX NORTH: William F. Ryan, Lowell, 219 Central Street. (Alternate: Walter L. Twarog, Lowell, 117 Durant Street.)
NORFOLK: Charles J. Kickham, Brookline. Office, Boston (15), 508 Commonwealth Avenue. (Alternate: Henry M. Emmons, Boston, 354 Commonwealth Avenue.)
WORCESTER NORTH: C. Bertram Gay, Fitchburg, 62 Day Street. (Alternate: John J. Curley, Leominster, 89 West Street.)

*Term Expires 1948*

ESSEX SOUTH: Walter G. Phippen, Salem, 31 Chestnut Street. (Alternate: None.)
HAMPSHIRE: Henry A. Tadgell, Belchertown, Belchertown State School. (Alternate: Lawrence N. Durgin, Amherst, 66 Amity Street.)
MIDDLESEX SOUTH: Harold G. Giddings, Newton Centre. Office, Boston (16), 270 Commonwealth Avenue. (Alternate: Arthur M. Jackson, Everett [49], 512 Broadway.)
NORFOLK SOUTH: Daniel B. Reardon, Quincy (69), 1186 Hancock Street. (Alternate: Nahum R. Pillsbury, South Braintree [85], Norfolk County Hospital.)
SUFFOLK: Alexander J. A. Campbell, Boston (15), 520 Commonwealth Avenue. (Alternate: Howard F. Root, Boston [15], 81 Bay State Road.)

WORCESTER: Bancroft C. Wheeler, Worcester, 27 Elm Street. (Alternate: Franklyn P. Bousquet, Worcester, 390 Main Street [interim appointment].)

*Term Expires 1949*

BARNSTABLE: Paul M. Butterfield, Harwich. (Alternate: Paul P. Henson, Hyannis, 149 Main Street.)

BRISTOL NORTH: Joseph L. Murphy, Taunton, 23 Cedar Street. (Alternate: Curtis B. Kingsbury, Taunton, 63 Prospect Street.)

BRISTOL SOUTH: Richard B. Butler, Fall River, 278 North Main Street. (Alternate: Curtis C. Tripp, New Bedford, 416 County Street.)

ESSEX NORTH: Rolf C. Norris, Methuen, 247 Broadway. (Alternate: George J. Connor, Haverhill, 81 Merrimack Street.)

MIDDLESEX EAST: Kenneth L. Maclachan, Melrose, 1 Bellevue Avenue. (Alternate: Justin L. Anderson, Reading, 53 Woburn Street.)

PLYMOUTH: George A. Moore, Brockton, 167 Newbury Street. (Alternate: Alfred L. Duncombe, Brockton, 38 Winthrop Street.)

**Committee on Public Relations** — Established 1931 (one councilor elected yearly by each district medical society; the president and president-elect of the Society are chairman and vice-chairman, respectively, and the vice-president and secretary of the Society are members ex-officiis).

BARNSTABLE: Paul P. Henson, Hyannis, 149 Main Street.

BERKSHIRE: Patrick J. Sullivan, Dalton, 471 Main Street.

BRISTOL NORTH: Milton E. Johnson, Attleboro, 33 Bank Street.

BRISTOL SOUTH: Harold E. Perry, New Bedford, 159 Cottage Street.

ESSEX NORTH: Harold R. Kurth, Lawrence, 57 Jackson Street.

ESSEX SOUTH: Loring Grimes, Swampscott, 84 Humphrey Street.

FRANKLIN: Frank A. Millett, Greenfield, 40 High Street.

HAMPDEN: Patrick E. Gear, Holyoke, 188 Chestnut Street.

HAMPSHIRE: Joseph R. Hobbs, Williamsburg, Main Street.

MIDDLESEX EAST: Milton J. Quinn, Winchester, 44 Church Street.

MIDDLESEX NORTH: Daniel J. Ellison, Lowell, 8 Merrimack Street.

MIDDLESEX SOUTH: Gordon M. Morrison, Waban. Office, Boston (15), 520 Commonwealth Avenue.

NORFOLK: Dean S. Luce, Canton, 553 Washington Street.

NORFOLK SOUTH: Henry A. Robinson, Hingham, 205 North Street.

PLYMOUTH: Charles D. McCann, Brockton, 12 Cottage Street.

SUFFOLK: Albert A. Hornor, Boston (15), 319 Longwood Avenue.

WORCESTER: Nicholas S. Scarcello, Worcester, 1 Sheldon Street.

WORCESTER NORTH: James V. McHugh, Leominster, 55 West Street.

**Subcommittees of the Committee on Public Relations**

LABOR AND INDUSTRY — Established 1945.

Daniel B. Reardon (Norfolk South), *chairman*; George J. Connor, Essex North; Daniel J. Ellison, Middlesex North; John Fallon, Worcester; and Michael A. Tighe, Middlesex North.

COMMITTEE TO MEET WITH THE MEDICAL ADVISORY COMMITTEE OF THE INDUSTRIAL ACCIDENT BOARD — Established 1942.

Daniel J. Ellison (Middlesex North), *chairman*; Gordon M. Morrison, Middlesex South; and David D. Scannell, Norfolk.

POSTPAYMENT MEDICAL CARE — Established 1942.

Daniel J. Ellison (Middlesex North), *chairman*; *Michael F. Barrett*, Plymouth; *James H. Brewster*, Bristol North; *James T. Brosnan*, Worcester; *Lucien R. Chaput*, Essex North; *Charles F. Fasce*, Berkshire; Patrick E. Gear, Hampden; Loring Grimes, Essex South; *Francis T. Janizen*, Suffolk; Egon E. Kattwinkel, Middlesex South; *Howard M. Kemp*, Franklin; *William B. LeBrecht*, Worcester North; *Wilfred L. McKenzie*, Middlesex East; Harold E. Perry, Bristol South; Daniel B. Reardon, Norfolk South; Harold F. Rowley, Barnstable; *Elmer E. Thomas*, Hampshire (interim appointment) and Norman A. Welch, Norfolk.

TAX-SUPPORTED MEDICAL CARE — Established 1940.

Thomas Hunter (Worcester), *chairman* (interim appointment); Frederick S. Hopkins, Hampden; Albert A. Hornor, Suffolk; *William J. Pelletier*, Franklin; and Frank W. Snow, Essex North.

**Committee on Legislation** — Established 1942 (one councilor elected yearly by each district medical society).

BARNSTABLE: To be appointed.

BERKSHIRE: John Hughes, Pittsfield, 74 North Street.

BRISTOL NORTH: Curtis B. Kingsbury, Taunton, 63 Prospect Street.

BRISTOL SOUTH: Curtis C. Tripp, New Bedford, 416 County Street.

ESSEX NORTH: Nicandro F. DeCesare, Methuen. Office, Lawrence, 57 Jackson Street.

ESSEX SOUTH: To be appointed.

FRANKLIN: Arthur W. Hayes, Greenfield, 78 Federal Street.

HAMPDEN: Arthur H. Riordan, Indian Orchard, 147 Oak Street.

HAMPSHIRE: Lawrence N. Durgin, Amherst, 66 Amity Street.

MIDDLESEX EAST: John M. Wilcox, Woburn, 6 Bennett Street.

MIDDLESEX NORTH: Archibald R. Gardner, Lowell, 16 Shattuck Street.

MIDDLESEX SOUTH: Kenneth J. Tillotson, Belmont. Office, Waverly, McLean Hospital.

NORFOLK: To be appointed.

NORFOLK SOUTH: David L. Belding, Hingham. Office, Boston (18), 80 East Concord Street.

PLYMOUTH: Alfred L. Duncombe, Brockton, 38 Winthrop Street.

SUFFOLK: William E. Browne, Boston (15), 587 Beacon Street.

WORCESTER: George R. Dunlop, Worcester, 53 Massachusetts Avenue.

WORCESTER NORTH: John J. Curley, Leominster, 89 West Street.

**Subcommittee of the Committee on Legislation**

NATIONAL LEGISLATION — Established 1946.

Elmer S. Bagnall, (Essex North), *chairman*; Reginald Fitz, Suffolk; *James C. McCann*, Worcester; and Michael A. Tighe, Middlesex North.

**Committee on Nominations** — Established 1874 (one councilor and alternate elected yearly by each district medical society).

BARNSTABLE: Paul M. Butterfield, Harwich. (Alternate: Paul P. Henson, Hyannis, 149 Main Street.)

BERKSHIRE: Patrick J. Sullivan, Dalton, 471 Main Street. (Alternate: John Hughes, Pittsfield, 78 North Street.)

BRISTOL NORTH: Joseph L. Murphy, Taunton, 23 Cedar Street. (Alternate: Curtis B. Kingsbury, Taunton, 63 Prospect Street.)

BRISTOL SOUTH: Edmond F. Cody, New Bedford, 105 South 6th Street. (Alternate: Richard B. Butler, Fall River, 278 North Main Street.)

ESSEX NORTH: Charles F. Warren, Amesbury, 155 Main Street. (Alternate: Percy J. Look, Andover, 115 Main Street.)

ESSEX SOUTH: Peer P. Johnson, Beverly, 1 Monument Square. (Alternate: De Witt S. Clark, Salem, 2 Oliver Street.)

FRANKLIN: John E. Moran, Greenfield, 31 Federal Street. (Alternate: Arthur W. Hayes, Greenfield, 78 Federal Street.)

HAMPDEN: Allen G. Rice, Springfield, 146 Chestnut Street. (Alternate: Harry F. Byrnes, Springfield, 6 Chestnut Street.)

HAMPSHIRE: Henry A. Tadgell, Belchertown, Belchertown State School. (Alternate: Lawrence N. Durgin, Amherst, 66 Amity Street.)

MIDDLESEX EAST: Ralph R. Stratton, Melrose (76), 538 Lynn Fells Parkway. (Alternate: Edward M. Halligan, Reading, 37 Salem Street.)

MIDDLESEX NORTH: Adam E. Shaw, Lowell, 386 Andover Street. (Alternate: Walter L. Twarog, Lowell, 117 Durant Street.)

*Interim appointment.

MIDDLESEX SOUTH: Harold G. Giddings, Newton Centre. Office, Boston, 270 Commonwealth Avenue. (Alternate Joseph C. Merriam, Framingham, 198 Union Avenue.)
NORFOLK: Albert Ehrenfried, Brookline. Office, Boston (15), 520 Commonwealth Avenue. (Alternate: Carlton E. Allard, Dorchester, 428 Columbia Road.)
NORFOLK SOUTH: Daniel B. Reardon, Quincy (69), 1186 Hancock Street. (Alternate: Frederick Hinchliffe, Cohasset, 117 South Main Street.)
PLYMOUTH: Bradford H. Peirce, South Hanson, Plymouth County Hospital. (Alternate: Alfred L. Duncombe, Brockton, 38 Winthrop Street.)
SUFFOLK: Albert A. Hornor, Boston (15), 319 Longwood Avenue. (Alternate: Conrad Wesselhoeft, Boston, 315 Marlborough Street.)
WORCESTER: Ralph S. Perkins, Worcester, 10 Hackfeld Road (Alternate: Franklyn P. Bousquet, Worcester [8], 390 Main Street [interim appointment]).
WORCESTER NORTH: John J. Curley, Leominster, 89 West Street. (Alternate: James V. McHugh, Leominster, 55 West Street.)

## STANDING COMMITTEES FOR 1946–1947

### ELECTED BY THE COUNCIL, MAY 21, 1946

| | *Date of Appointment* |
|---|---|
| **ARRANGEMENTS — Established 1849.** | |
| Sidney C. Wiggin, Suffolk | June 24, 1942 (appointed chairman May 21, 1946) |
| *G. Guy Bailey, Jr.*,* Middlesex South | November 13, 1942 |
| John F. Conlin, Suffolk | May 21, 1946 |
| Harold G. Giddings, Middlesex South | May 22, 1944 |
| Robert L. Goodale, Suffolk | May 22, 1944 |
| **ETHICS AND DISCIPLINE — Established 1871.** | |
| Ralph R. Stratton, Middlesex East | June 9, 1936 (appointed chairman May 21, 1941) |
| William J. Brickley, Suffolk | February 3, 1937 |
| Archibald R. Gardner, Middlesex North | May 21, 1941 |
| Fred R. Jouett, Middlesex South | May 21, 1940 |
| Allen G. Rice, Hampden | June 1, 1938 |
| **FINANCE — Established 1938.** | |
| Robert W. Buck, Middlesex South | May 21, 1946 (chairman) |
| *Francis C. Hall*, Suffolk | July 8, 1943 |
| Fabyan Packard Middlesex South | May 21, 1946 |
| Bancroft C. Wheeler, Worcester | May 21, 1946 |
| Charles F. Wilinsky, Suffolk | June 2, 1938 |
| **INDUSTRIAL HEALTH — Established 1942.** | |
| Daniel L. Lynch, Norfolk | May 25, 1942 (appointed chairman May 21, 1946) |
| *Joseph C. Aub*, Suffolk | May 25, 1942 |
| *Louis R. Daniels*, Middlesex South | May 22, 1944 |
| John G. Downing, Middlesex South | May 22, 1944 |
| Patrick E. Gear, Hampden | May 23, 1945 |
| Harold R. Kurth Essex North | May 23, 1945 |
| *Thomas L. Shipman*, Essex South | May 25, 1942 |
| **ADVISORY COMMITTEE TO COMMITTEE ON INDUSTRIAL HEALTH — Established 1942.** | |
| *Philip Drinker* | |
| *Harriet L. Hardy* | |
| *Emma S. Tousant* | |
| **MEDICAL DEFENSE — Established 1927.** | |
| Arthur W. Allen, Suffolk | June 7, 1927 (appointed chairman June 7, 1939) |
| Edwin D. Gardner, Bristol South | June 7, 1927 |
| Charles J. Kickham, Norfolk | May 21, 1946 |
| *William R. Morrison*, Suffolk | June 9, 1936 |
| *Horatio Rogers*, Suffolk | June 7, 1939 |
| **MEDICAL EDUCATION — Established 1881.** | |
| Robert T. Monroe, Norfolk | May 21, 1941 (appointed chairman February 4, 1942) |
| *James M. Faulkner*, Norfolk | May 21, 1946 |
| George D. Henderson, Hampden | June 1, 1938 |
| Isaac R. Jankelson, Norfolk | May 25, 1942 |
| Chester S. Keefer, Suffolk | February 4, 1942 |
| **MEMBERSHIP — Established 1897.** | |
| Peirce H. Leavitt, Plymouth | June 1, 1938 (appointed chairman [interim appointment] September 3, 1946) |
| William A. R. Chapin, Hampden | May 23, 1945 |
| Lewis S. Pilcher, Middlesex South (interim appointment) | July 26, 1946 |
| Guy L. Richardson, Essex North | May 21, 1946 |
| Samuel N. Vose, Suffolk | March 15, 1944 |
| H. Quimby Gallupe (Middlesex South), Albert E. Parkhurst (Essex South), William F. Ryan (Middlesex North) — representing the Supervising Censors. | |
| **PUBLICATIONS — Established 1825.** | |
| Richard M. Smith, Suffolk | June 6, 1933 (appointed chairman May 21, 1941) |
| *Oliver Cope*, Middlesex South | May 21, 1941 |
| John Fallon, Worcester | November 14, 1944 |
| James P. O'Hare, Suffolk | June 9, 1936 |
| Conrad Wesselhoeft, Suffolk | June 2, 1937 |
| **PUBLIC HEALTH — Established 1912.** | |
| Roy J. Ward, Worcester | May 22, 1944 (chairman) |
| Elmer S. Bagnall, Essex North | May 23, 1945 |
| *John J. Poutas*, Middlesex South | May 21, 1946 |
| George L. Steele, Hampden | May 23, 1945 |
| Conrad Wesselhoeft, Suffolk | July 27, 1944 |
| **SOCIETY HEADQUARTERS — Established 1942.** | |
| Frank R. Ober, Suffolk | May 22, 1944 (appointed chairman November 1, 1944) |
| Albert A. Hornor, Suffolk | November 6, 1944 |
| Walter G. Phippen, Essex South | May 21, 1946 |
| Daniel B. Reardon, Norfolk South | May 22, 1944 |
| Michael A. Tighe, Middlesex North | May 24, 1943 |

*Italics indicate that the physician is not a member of the Council.

## SPECIAL COMMITTEES FOR 1946–1947

### ELECTED BY THE COUNCIL, MAY 21, 1946

**COMMITTEE ON CANCER — Established 1917.**
George A. Moore (Plymouth), *chairman*; *Thomas J. Anglem*, Suffolk; *Ernest M. Daland*, Suffolk; Allen G. Rice, Hampden; and *Channing C. Simmons*, Suffolk.

**COMMITTEE ON COUNCIL RULES — Established 1944.**
Charles E. Mongan (Middlesex South), *chairman*; Elmer S. Bagnall, Essex North; Frank R. Ober, Suffolk; George L. Schadt, Hampden; and Michael A. Tighe, Middlesex North.

**COMMITTEE TO CONSIDER EXPERT TESTIMONY — Established 1936.**
Frank R. Ober (Suffolk), *chairman*; Carl Bearse, Norfolk; William J. Brickley, Suffolk; David Cheever, Suffolk; and Francis P. McCarthy, Norfolk.

**COMMITTEE ON MATERNAL WELFARE — Established 1941.**
Raymond S. Titus (Norfolk), *chairman*; James M. Baty, Middlesex South; *Ralph E. Cole*, Middlesex North; *Arthur F. G. Edgelow*, Hampden; *Florence L. McKay*, Suffolk; Joseph C. Merriam, Middlesex South; and Louis E. Phaneuf, Suffolk.

COMMITTEE TO MAKE A SURVEY OF MALPRACTICE INSURANCE IN MASSACHUSETTS — Established 1946.

Carl Bearse (Norfolk), *chairman*; William J. Brickley, Suffolk; Edwin D. Gardner, Bristol South; Daniel B. Reardon, Norfolk South; and Guy L. Richardson, Essex North.

COMMITTEE TO ASSIST THE COUNCIL ON MEDICAL EDUCATION AND HOSPITALS OF THE AMERICAN MEDICAL ASSOCIATION IN THE PROVISIONAL APPROVAL OF CERTAIN MASSACHUSETTS HOSPITALS — Established 1946.

Robert T. Monroe (Norfolk), *chairman*; H. Quimby Gallupe, Middlesex South; Walter G. Phippen, Essex South; Michael A. Tighe, Middlesex North; and Charles F. Wilinsky, Suffolk.

MEDICAL ADVISORY COMMITTEE TO REGIONAL OPA — Established 1943.

Joseph Garland (Suffolk), *chairman*; *F. Gorham Brigham*, Norfolk; Clifford L. Derick, Middlesex South; Loring Grimes, Essex South; and *Franklin W. White*, Suffolk.

COMMITTEE ON PHYSICAL MEDICINE — Established 1945.

Arthur L. Watkins (Middlesex South), *chairman*: *Ralph M. Chambers*, Bristol North; *Franklin P. Lowry*, Middlesex South; *Robert B. Osgood*, Suffolk; and Henry A. Tadgell, Hampshire.

COMMITTEE ON POSTWAR LOAN FUND — Established 1945.

George L. Schadt (Hampden), *chairman*; Eliot Hubbard, Jr., Middlesex South; Charles C. Lund, Suffolk; Albert E. Parkhurst, Essex South; and Michael A. Tighe, Middlesex North.

COMMITTEE ON POSTWAR PLANNING — Established 1944.

Howard F. Root (Suffolk), *chairman*; Elmer S. Bagnall, Essex North; George Ballantyne, Worcester; Vlado A. Getting, Middlesex South; Charles J. Kickham, Norfolk; Kenneth L. Maclachlan, Middlesex East; *Leland S. McKittrick*, Suffolk; Robert N. Nye, Suffolk; W. Richard Ohler, Norfolk; Leroy E. Parkins, Suffolk; and Walter G. Phippen, Essex South.

**Subcommittees of the Committee on Postwar Planning**

CO-ORDINATING ON EDUCATION — Established 1944.

*Charles F. Branch*, Suffolk; Nathaniel W. Faxon, Suffolk; and W. Richard Ohler, Norfolk.

HOSPITALS — Established 1944.

Charles F. Wilinsky (Suffolk), *chairman*; Arthur W. Allen, Suffolk; Nathaniel W. Faxon, Suffolk; *Harold Jeghers*, Norfolk; *James W. Manary*, Suffolk; and *Clifton T. Perkins*, Middlesex East.

MEDICAL ECONOMICS — Established 1944.

Leland S. McKittrick (Suffolk), *chairman*; Elmer S. Bagnall, Essex North; Allan M. Butler, Suffolk; Vlado A. Getting, Middlesex South; and Merrill C. Sosman, Suffolk.

MEDICAL SCHOOLS — Established 1944.

*Charles F. Branch* (Suffolk), *chairman*; *C. Sidney Burwell*, Norfolk; and Walter G. Phippen, Essex South.

ORGANIZATION — Established 1944.

Walter G. Phippen (Essex South), *chairman*; George Ballantyne, Worcester; Frederick S. Hopkins, Hampden; *Chester M. Jones*, Suffolk; Frank R. Ober, Suffolk; *Joseph W. O'Connor*, Worcester; *Francis J. Steele*, Worcester; and Michael A. Tighe, Middlesex North.

POSTGRADUATE MEDICAL EDUCATION — Established 1944.

W. Richard Ohler (Norfolk), *chairman*; Vlado A. Getting, Middlesex South; *G. Philip Grabfield*, Suffolk; Lewis M. Hurxthal, Suffolk; Charles J. Kickham, Norfolk; *Eugene M. Landis*, Middlesex South; *Charles G. Mixter*, Suffolk; Robert N. Nye, Suffolk; *Joseph W. O'Connor*, Worcester; *Samuel H. Proger*, Norfolk; and Harry C. Solomon, Suffolk.

COMMITTEE ON VETERANS' AFFAIRS — Established 1946.

*G. Philip Grabfield* (Suffolk), *chairman*; *Victor G. Balboni*, Suffolk; *George P. Denny*, Suffolk; *Alexander Marble*, Suffolk; and *George F. Wilkins*, Middlesex South.

In addition the following are representatives from the various district medical societies:

*Charles Bradford*,* Suffolk
*Stephen Brown*,*Hampshire
*William M. Carr*,* Plymouth
*Leo R. Desmond*,* Norfolk
*Spencer C. Flo*,* Franklin
*Merrill F. Gardner*,* Bristol South
*Willis M. Gowen*,* Middlesex East
*Leonard W. Hill*,* Bristol North
*Sheldon L. Hunt*,* Barnstable
Thomas Hunter,* Worcester
*John C. McGirr*,* Middlesex South
*Franklin K. Paddock*,* Berkshire
*Ensio K. F. Ronka*,* Norfolk South
*Louis B. Simard*,* Essex North
*Thomas J. G. Tighe*,* Middlesex North

AUDITING COMMITTEE.

*Howard B. Jackson* (Norfolk), *chairman*; and *Frank T. Downey*, Middlesex South (interim appointment).

COMMITTEE ON POSTGRADUATE ASSEMBLY — Established 1946.

Leroy E. Parkins (Suffolk), *chairman*; Harold G. Giddings, Middlesex South; Frederick S. Hopkins, Hampden; Charles J. Kickham, Norfolk; and Robert N. Nye, Suffolk.

COMMITTEE ON REHABILITATION — Established 1941.

Joseph H. Shortell (Suffolk), chairman; *Benjamin F. Andrews*, Worcester; *Ralph M. Chambers*, Bristol North; *William M. Collins*, Middlesex North; *James J. Regan*, Suffolk; and Arthur L. Watkins, Middlesex South.

COMMITTEE TO MAKE RECOMMENDATIONS AS TO FUTURE DIRECTORS OF THE BLUE SHIELD — Established 1945.

Leland S. McKittrick (Suffolk), *chairman* (term expires May 1949); Harold G. Giddings, Middlesex South (term expires May, 1951); *Elliott P. Joslin*, Suffolk (term expires May, 1947); Peirce H. Leavitt, Plymouth (term expires May, 1950); and *George Gilbert Smith*, Suffolk (term expires May, 1948).

COMMITTEE TO MEET WITH THE MASSACHUSETTS HOSPITAL ASSOCIATION — Established 1940.

Walter G. Phippen (Essex South), *chairman*; *Edward A. Adams*, Worcester North; Nicholas S. Scarcello, Worcester (interim appointment); Edwin D. Gardner, Bristol South; Frederic Hagler, Hampden; and Albert E. Parkhurst, Essex South.

COMMITTEE TO CONFER WITH THE MASSACHUSETTS FARM BUREAU FEDERATION — Established 1945.

Joseph C. Merriam (Middlesex South), *chairman*: John E. Moran, Franklin; and *Elmer E. Thomas*, Hampshire (interim appointment).

COMMITTEE TO SURVEY SALARIES — Established 1946.

Charles J. Kickham (Norfolk), *chairman* (interim appointment); Robert W. Buck, Middlesex South (interim appointment); and Robert N. Nye, Suffolk (interim appointment).

COMMITTEE TO STUDY INCREASE IN ASSESSMENT OF DUES — Established 1946.

Frank R. Ober (Suffolk), *chairman* (interim appointment); N. Newall Copeland, Berkshire (interim appointment); Edwin D. Gardner, Bristol South (interim appointment); Kenneth L. Maclachlan, Middlesex East (interim appointment); and *John W. McKoan*, Worcester (interim appointment).

*Interim appointment.

COMMITTEE TO STUDY INCOME LEVEL FOR BLUE SHIELD — Established 1946.

Charles F. Wilinsky (Suffolk), *chairman* (interim appointment); *Norman H. Bruce*, Middlesex South (interim appointment); Raoul L. Drapeau, Middlesex North (interim appointment); Henry L. Kirkendall, Worcester (interim appointment); and John W. Spellman, Norfolk (interim appointment).

COMMITTEE OF SEVEN — Established 1946.

Elmer S. Bagnall, Essex North; David Cheever, Suffolk; Roger I. Lee, Suffolk; Charles E. Mongan, Middlesex South; Frank R. Ober, Suffolk; Walter G. Phippen, Essex South; and George L. Schadt, Hampden.

COMMITTEE TO MEET WITH GENERAL HAWLEY WITH VIEW OF FORMULATING PROGRAM IN MASSACHUSETTS FOR MEDICAL CARE OF VETERANS AND THEIR DEPENDENTS — Established 1945.

Humphrey L. McCarthy (Norfolk), *chairman*; *James K. Bragger*, Norfolk (interim appointment); and Michael A. Tighe, Middlesex North.

REPRESENTATIVES TO THE MASSACHUSETTS CENTRAL HEALTH COUNCIL.

George D. Henderson (Hampden), *chairman*; Elmer S. Bagnall, Essex North; *James W. Bunce*, Berkshire; *Merrill E. Champion*, Suffolk; and *Robert B. Osgood*, Suffolk.

REPRESENTATIVE TO THE HOSPITAL COUNCIL OF BOSTON FOR THE YEAR 1946.

William E. Browne, Suffolk.

REPRESENTATIVE ON THE LEGISLATIVE COMMITTEE OF THE MASSACHUSETTS CENTRAL HEALTH COUNCIL.

William E. Browne, Suffolk.

REPRESENTATIVE ON A PROFESSIONAL ADVISORY COMMITTEE ORGANIZED BY THE DIVISION OF VOCATIONAL REHABILITATION OF STATE DEPARTMENT OF EDUCATION FOR PURPOSE OF ESTABLISHING A PROGRAM ON PHYSICAL RESTORATION.

*Joseph H. Shortell*, Suffolk.

REPRESENTATIVES TO THE COUNCIL OF THE NEW ENGLAND STATE MEDICAL SOCIETIES.

Allen G. Rice, Hampden; Michael A. Tighe, Middlesex North; and Norman A. Welch, Norfolk (interim appointment).

REPRESENTATIVE FOR SURVEY BY ACADEMY OF PEDIATRICS. *Gerald N. Hoeffel*, Middlesex South (interim appointment).

TWENTY-FIVE VOTING MEMBERS IN MASSACHUSETTS HOSPITAL SERVICE, INC.

*Benjamin H. Alton*, Worcester; *Gerardo M. Balboni*, Suffolk; *Laurence D. Chapin*, Hampden; *Lucien R. Chaput*, Essex North; Hilbert F. Day, Middlesex South; *George K. Fenn*, Essex South; *Joseph E. Flynn*, Middlesex South; Archibald R. Gardner, Middlesex North; Harold G. Giddings, Middlesex South; Henry W. Godfrey, Middlesex South; Albert A. Hornor, Suffolk; *John H. Lambert*, Middlesex North; Alexander A. Levi, Middlesex South; Raymond A. McCarthy, Middlesex South; Joseph C. Merriam, Middlesex South; Donald Munro, Suffolk, Albert E. Parkhurst, Essex South; Lewis S. Pilcher, Middlesex South; Helen S. Pittman, Suffolk; Allen G. Rice, Hampden; *Arthur T. Ronan*, Norfolk; George L. Steele, Hampden;Ralph R. Stratton, Middlesex East; *John E. Talbot*, Worcester; and *Edward L. Young*, Norfolk.

## DELEGATES AND ALTERNATES TO THE HOUSE OF DELEGATES OF THE AMERICAN MEDICAL ASSOCIATION FOR 1946–1947

| DELEGATES | ALTERNATES |
|---|---|
| *June 1, 1945, to June 1, 1947* | |
| Charles J. Kickham, Norfolk | John Fallon, Worcester |
| *Leland S. McKittrick*, Suffolk | Patrick J. Sullivan, Berkshire |
| *June 1, 1946, to June 1, 1948* | |
| David D. Scannell, Norfolk | Elmer S. Bagnall, Essex North |
| Dwight O'Hara, Middlesex South | *William J. Pelletier*, Franklin |
| Charles E. Mongan, Middlesex South | Patrick E. Gear, Hampden |
| Walter G. Phippen, Essex South | *John I. B. Vail*, Barnstable |

## COUNCILORS FOR 1946–1947

(ELECTED BY THE DISTRICT MEDICAL SOCIETIES AT THEIR ANNUAL MEETINGS, APRIL 15 TO MAY 15, 1946.)

BARNSTABLE

H. F. Rowley, Harwich Port, V. P.
P. M. Butterfield, Harwich, E. C., M. N. C.
P. P. Henson, Hyannis, 149 Main St., A. E. C., A. M. N. C., P. R. C.
C. H. Keene, Chatham, Seaview St.
O. S. Simpson, Centreville, Main St., Sec.

BERKSHIRE

N. N. Copeland, Pittsfield, 131 North St., V. P.
D. N. Beers, Pittsfield, 74 North St., Sec.
I. S. F. Dodd, Pittsfield, 34 Fenn St., Vice-President, E. C.
John Hughes, Pittsfield, 74 North St., A. M. N. C., Leg. C.
C. F. Kernan, Pittsfield, 184 North St., A. E. C.
Solomon Schwager, Pittsfield, 246 North St.
Helen M. Scoville, Pittsfield, House of Mercy Hospital.
P. J. Sullivan, Dalton, 471 Main St., M. N. C., P. R. C.
E. R. Wyman, Great Barrington, 259 Main St.

BRISTOL NORTH

W. M. Stobbs, Attleboro, 63 Bank St., V. P.
W. H. Allen, Mansfield, 70 North Main St.
M. E. Johnson, Attleboro, 33 Bank St., P. R. C.
C. B. Kingsbury, Taunton, 63 Prospect St., A. E. C., A. M. N. C., Leg. C.
W. J. Morse, Attleboro, 34 Sanford St., Sec.
J. L. Murphy, Taunton, 23 Cedar St., E. C., M. N. C.

BRISTOL SOUTH

S. P. Wilde, New Bedford, 73 Borden St., V. P.
R. B. Butler, Fall River, 278 North Main St., E. C., A. M. N. C.
E. F. Cody, New Bedford, 105 South Sixth St., M. N. C.
J. C. Corrigan, Fall River, 422 North Main St.
J. E. Fell, Fall River, 181 Purchase St., Sec.
J. A. Fournier, Fall River, 11 Choate St.
E. D. Gardner, Marion, Box 175.
R. H. Goodwin, New Bedford, 15 South 6th St.
William Mason, Fall River, 151 Rock St.
D. R. Mills, Edgartown, Pease Point Way.
H. E. Perry, New Bedford, 159 Cottage St., P. R. C.
C. C. Tripp, New Bedford, 416 County St., A. E. C., Leg. C.
Henry Wardle, Fall River, 173 Purchase St.

ESSEX NORTH

G. L. Richardson, Haverhill, 94 Emerson St., V. P.
E. S. Bagnall, Groveland, 281 Main St., Ex-Pres.
R. V. Baketel, Methuen, 7 Hampshire St.
R. E. Blais, Amesbury, 165 Main St.
G. J. Connor, Haverhill, 81 Merrimack St., A. E. C.
Elizabeth Councilman, Newburyport, 83 High St.
N. F. DeCesare, Lawrence, 57 Jackson St., Leg. C.
H. A. Fenton, Lawrence, 36 Winthrop Ave.
H. R. Kurth, Lawrence, 57 Jackson St., Sec., P. R. C.
P. J. Look, Andover, 115 Main St., A. M. N. C.
R. J. Neil, Methuen, 255 Broadway.
R. C. Norris, Methuen, 247 Broadway, E. C.
F. W. Snow, Newburyport, 24 Essex St.
C. F. Warren, Amesbury, 1 School St., M. N. C.

ESSEX SOUTH

C. A. Worthen, Lynn, 19 Park St., V. P.
K. J. Chadwell, Swampscott, 26 Lexington Circle.
D. S. Clark, Salem, 2 Oliver St., A. M. N. C.
R. E. Foss, Peabody, 125 Main St.
Loring Grimes, Swampscott, 84 Humphrey St., P. R. C.
C. A. Herrick, Manchester, 21 Union St.
W. R. Irving, Gloucester, 35 Middle St.
P. P. Johnson, Beverly, 1 Monument Sq., M. N. C.
H. M. Lowd, Swampscott, 90 Burill St.

B. B. Mansfield, Ipswich, 4 Green St.
A. E. Parkhurst, Beverly, 1 Monument Sq.
O. S. Pettingill, Middleton, Essex Sanatorium.
W. G. Phippen, Salem, 31 Chestnut St., Ex-Pres., E. C.
E. D. Reynolds, Danvers, 48 High St.
J. R. Shaughnessey, Salem, 24½ Winter St.
H. D. Stebbins, Salem, 342 Essex St., Sec.
P. E. Tivnan, Salem, 70 Washington St.
C. F. Twomey, East Lynn, 80 Ocean St.

FRANKLIN

J. B. Temple, Shelburne Falls, 11 Main St., V. P.
H. L. Craft, Ashfield, Sec.
A. W. Hayes, Greenfield, 78 Federal St., A. M. N. C., Leg. C.
F. A. Millett, Greenfield, 40 High St., A. E. C., P. R. C.
J. E. Moran, Greenfield, 31 Federal St., E. C., M. N. C.

HAMPDEN

Adolph Franz, Jr., Holyoke, 1158 Northampton St., V. P.
F. H. Allen, Holyoke, 16 Fairfield St.
E. P. Bagg, Holyoke, 207 Elm St., President-Elect.
J. M. Birnie, Springfield, 146 Chestnut St., Ex-Pres.
H. F. Byrnes, Springfield, 6 Chestnut St., A. M. N. C.
W. A. R. Chapin, Springfield, 121 Chestnut St., E. C.
J. L. Chereskin, Springfield, 333 Bridge St.
A. J. Douglas, Westfield, 30 Court St., A. E. C.
E. C. Dubois, Springfield, 174 Buckingham St.
P. E. Gear, Holyoke, 188 Chestnut St., P. R. C.
Frederic Hagler, Springfield, 20 Maple St.
G. D. Henderson, Holyoke, 176 Chestnut St.
F. S. Hopkins, Springfield, 146 Chestnut St.
Charles Jurist, Springfield, 70 Chestnut St.
M. W. Pearson, Ware, 19 Pleasant St.
A. G. Rice, Springfield, 146 Chestnut St., M. N. C.
A. H. Riordan, Indian Orchard, 147 Oak St., Leg. C.
G. L. Schadt, Springfield, 44 Chestnut St., Ex-Pres.
J. A. Seaman, Longmeadow, Office Springfield, 20 Maple St.
G. C. Steele, West Springfield, 39 Upper Church St., Sec.
G. L. Steele, Springfield, 20 Maple St.
W. W. Teahan, Holyoke, 217 Essex St.

HAMPSHIRE

J. J. Curran, Northampton, 16 Centre St., V. P.
L. N. Durgin, Amherst, 66 Amity St., A. E. C., A. M. N. C., Leg. C.
J. R. Hobbs, Williamsburg, Main St., P. R. C.
L. B. Pond, Easthampton, 115 Main St.
Mary P. Snook, Worthington, Sec.
H. A. Tadgell, Belchertown, Belchertown State School, E. C., M. N. C.

MIDDLESEX EAST

W. H. Flanders, Melrose 76, 481 Lebanon St., V. P.
J. L. Anderson, Reading, 53 Woburn St., A. E. C.
Robert Dutton, Wakefield, 33 Avon St.
E. M. Halligan, Reading, 37 Salem St., A. M. N. C.
R. W. Layton, Melrose 76, 8 Porter St., Sec.
K. L. Maclachlan, Melrose 76, 1 Bellevue Avenue, E. C.
M. J. Quinn, Winchester, 44 Church St., P. R. C.
W. F. Regan, Winchester, 101 High St.
R. R. Stratton, Melrose 76, 538 Lynn Fells Parkway, M. N. C., C.
J. M. Wilcox, Woburn, 6 Bennett St., Leg. C.

MIDDLESEX NORTH

W. F. Ryan, Lowell, 219 Central St., V. P., E. C.
R. L. Drapeau, Dracut, 1346 Bridge St.
D. J. Ellison, Lowell, 8 Merrimack St., P. R. C.
A. R. Gardner, Lowell, 16 Shattuck St., Leg. C.
S. G. Hajjar, North Billerica, 2 Talbot Ave.
B. D. Leahey, Lowell, 9 Central St., Sec.
A. E. Shaw, Lowell, 386 Andover St., M. N. C.
M. A. Tighe, Lowell, 9 Central St., Sec.
W. L. Twarog, Lowell, 117 Durant St., A. E. C., A. M. N. C.

MIDDLESEX SOUTH

J. F. Casey, Allston, Office Boston 15, 475 Commonwealth Ave., V. P.
E. W. Barron, Malden 48, Office Boston, 20 Ash St.
Harris Bass, Everett 49, 351 Broadway.
J. M. Baty, Belmont, Office Brookline 46, 1101 Beacon St.
J. D. Bennett, West Somerville 44, 72 College Ave.
E. H. Bigelow, Framingham Centre, 65 Edgell Rd., Ex-Pres.
W. O. Blanchard, Newton 58, 465 Centre St.
G. F. H. Bowers, Newton Highlands 61, 156 Woodward St.
Alice M. Broadhurst, Watertown 72, 259 Mt. Auburn St.
Madelaine R. Brown, Cambridge, Office Boston 16, 264 Beacon St.
R. N. Brown, Malden 48, 621 Main St.
R. W. Buck, Waban, Office Boston 15, 5 Bay State Rd., C.
E. J. Butler, Cambridge, 25 Garden St.
C. W. Clark, Newtonville 60, 363 Walnut St.
J. A. Daley, Natick, 36 Pond St.
H. F. Day, Cambridge, 34 Kirkland St.
C. L. Derick, Newton Highlands, Office Boston 15, 412 Beacon St.
J. G. Downing, Newton, Office Boston 15, 520 Commonwealth Ave.
C. W. Finnerty, West Somerville 44, 440 Broadway.
H. Q. Gallupe, Waltham 54, 751 Main St.
F. W. Gay, Malden 48, 20 Park St.
V. A. Getting, Belmont, Office Boston 8, 546 State House.
H. G. Giddings, Newton Centre, Office Boston 16, 270 Commonwealth Ave., E. C., M. N. C.
H. W. Godfrey, Auburndale 66, 14 Hancock St.
J. L. Golden, Medford 55, 86 Forest St.
A. D. Guthrie, Medford 55, 408 Salem St.
Eliot Hubbard, Jr., Cambridge, 29 Highland St., Treasurer.
A. M. Jackson, Everett 49, 512 Broadway, A. E. C.
F. R. Jouett, Cambridge, 1 Craigie St.
E. E. Kattwinkel, West Newton 65, 65 Sterling St.
A. A. Levi, Newton, Office Boston 15, 481 Beacon St., Sec.
A. N. Makechnie, Cambridge, 14 Upland St.
R. A. McCarty, Waltham 54, 465 Lexington St.
P. H. Means, Cambridge, 1 Waterhouse St.
J. C. Merriam, Framingham, 198 Union Ave., A. M. N. C.
Dudley Merrill, Cambridge, 51 Brattle St.
C. E. Mongan, Somerville 43, 24 Central St., Ex-Pres.
G. M. Morrison, Waban, Office Boston 15, 520 Commonwealth Ave., P. R. C.
J. P. Nelligan, Cambridge, 2336 Massachusetts Ave.
E. J. O'Brien, Jr., Newton, Office Boston 16, 270 Commonwealth Ave.
Dwight O'Hara, Waltham, Office Boston 15, 416 Huntington Ave., President.
Fabyan Packard, Belmont, Office Boston, Soldiers' Field.
L. G. Paul, Newton Centre, Office Boston 16, 270 Commonwealth Ave.
L. S. Pilcher, Newton Centre 59, 43 Parker St.
Randolph Piper, Concord, 14 Sudbury Rd.
T. E. Reilly, Marlboro, 6 Newton St.
S. H. Remick, Waltham 54, 735 Trapelo Rd.
Max Ritvo, Newton, Office Boston 15, 416 Marlborough St.
E. H. Robbins, Somerville 43, 334 Broadway.
M. J. Schlesinger, Newton, Office Boston Boston 15, 330 Brookline Ave.
E. W. Small, Belmont 78, 68 Leonard St.
H. P. Stevens, Cambridge, 1 Craigie St.
K. J. Tillotson, Waverley 79, McLean Hospital, Leg. C.
A. B. Toppan, Watertown 72, 289 Mt. Auburn St.
J. E. Vance, Natick, Office Boston 15, 29 Bay State Rd.
Fresenius Van Nüys, Weston 93, 338 Boston Post Rd.
C. F. Walcott, Cambridge, 81 Sparks St.
A. L. Watkins, Arlington, Office Boston 14, Massachusetts General Hospital.
B. M. Wein, Newton, Office Boston 15, 471 Commonwealth Ave.
B. S. Wood, Weston, Office Waltham 54, 751 Main St.
Alfred Worcester, Waltham 54, 314 Bacon St., Ex-Pres.
Hovhannes Zovickian, Watertown 72, 528 Mt. Auburn St.

NORFOLK

J. H. Cauley, Dorchester, 8 Carruth St., V. P.
A. A. Abrams, Brookline 46, 153 Dean Rd.

C. E. Allard, Dorchester, 428 Columbia Rd., A. M. N. C.
B. E. Barton, West Roxbury 32, 10 Richwood St., Sec.
Carl Bearse, Boston 15, 483 Beacon St.
Arthur Berk, Brookline, Office Boston 16, 270 Commonwealth Ave.
D. J. Collins, Norwood, 100 Day St.
William Dameshek, Brookline, Office Boston 15, 113 Bay State Rd.
G. L. Doherty, West Roxbury, Office Boston 15, 466 Commonwealth Ave.
Albert Ehrenfried, Brookline, Office Boston 15, 520 Beacon St., M. N. C.
H. M. Emmons, Needham, Office Boston 15, 354 Commonwealth Ave.
Susannah Friedman, Roxbury, Office Boston 15, 485 Commonwealth Ave.
B. A. Godvin, Jamaica Plain, Office Boston 15, 483 Beacon St.
D. L. Halbersleben, Brookline 46, 42 Goodnough Rd.
J. B. Hall, Roxbury 19, 108 Dudley St.
H. B. Harris, East Milton, Office Dorchester, 487 Columbia Rd.
R. J. Heffernan, Jamaica Plain, Office Brookline 46, 1101 Beacon St.
P. J. Jakmauh, Milton, Office South Boston 27, 509 Broadway.
I. R. Jankelson, Jamaica Plain, Office Boston 15, 483 Beacon St.
C. J. Kickham, Brookline, Office Boston 15, 508 Commonwealth Ave., E. C.
C. J. E. Kickham, Jamaica Plain, Office Brookline 46, 1101 Beacon St.
D. L. Lionberger, Dedham, 709 East St.
D. S. Luce, Canton, 553 Washington St., P. R. C.
C. M. Lydon, Dorchester, 276 Bowdoin St.
D. L. Lynch, Roslindale, Office Boston, 245 State St., C.
F. P. McCarthy, Milton, Office Boston 15, 371 Commonwealth Ave.
H. L. McCarthy, West Roxbury, Office Boston 15, 479 Beacon St.
R. T. Monroe, Brookline, Office Boston 16, 270 Commonwealth Ave., C.
F. J. Moran, Dedham, 395 Washington St.
Hyman Morrison, Brookline, Office Boston 15, 483 Beacon St.
D. J. Mullane, Brookline 46, 1101 Beacon St.
J. J. O'Connell, Dorchester, 1061 Dorchester Ave.
W R. Ohler, Jamaica Plain, Office Boston 15, 319 Longwood Ave.
G. W. Papen, Brookline, Office Boston, 31 Milk St.
H. C. Petterson, West Roxbury, Office Boston 15, 29 Bay State Rd.
Frederick Reis, Jamaica Plain, Office Boston 15, 416 Huntington Ave.
H. A. Rice, Canton, 742 Washington St.
S. A. Robins, Boston 15, 636 Beacon St.
D. D. Scannell, Jamaica Plain, Office Boston 15, 475 Commonwealth Ave.
J. A. Seth, Milton, Office Boston 15, 47 Bay State Rd.
J. A. Sieracki, Norwood, 71 Winter St.
S. L. Skvirsky, Chestnut Hill, Office Boston, 336 State House.
E. C. Smith, Brookline, Office Boston 15, 520 Commonwealth Ave.
Kathleyne S. Snow, Jamaica Plain, Office Boston 15, 466 Commonwealth Ave.
J. W. Spellman, Chestnut Hill, Office Brookline 46, 1101 Beacon St.
M. H. Spellman, Jamaica Plain, Office Boston 15, 475 Commonwealth Ave.
A. R. Stagg, Medford, 25 Pleasant St.
J. P. Treanor, Jr., Jamaica Plain, Office Brookline 46, 1101 Beacon St.
W. J. Walton, Dorchester, 106 Bowdoin St.
N. A. Welch, West Roxbury, Office Boston 15, 520 Commonwealth Ave., Assistant Treasurer.
P. R. Withington, Milton 86, 350 Randolph Ave.
Louis Wolff, Brookline, Office Boston 16, 270 Commonwealth Ave.
E. T. Wyman, Brookline, Office Boston 15, 319 Longwood Ave.

NORFOLK SOUTH

D. L. Belding, Hingham, Office Boston 18, 80 East Concord St., V. P., Leg. C.
F. A. Bartlett, Wollaston 90, 308 Beale St. (interim appointment)
Harry Braverman, Quincy 69, 43 School St.
W. R. Helfrich, Quincy 69, 166 Washington St.
Frederick Hinchliffe, Cohasset, 117 South Main St., A. M. N. C.
E. K. Jenkins, South Braintree 85, Norfolk County Hospital, Sec.
N. R. Pillsbury, South Braintree 85, Norfolk County Hospital, A. E. C.
D. B. Reardon, Quincy 69, 1186 Hancock St., E. C., M. N. C.
H. A. Robinson, Hingham, 205 North St., P. R. C.
R. G. Vinal, Norwell, Main St.

PLYMOUTH

W. H. Pulsifer, Whitman, 26 Park Ave., V. P.
J. C. Angley, Bryantville, School St.
A. L. Duncombe, Brockton, 38 Winthrop St., A. E. C., A. M. N. C., Leg. C.
H. H. Hamilton, Plymouth, 70 Court St.
P. H. Leavitt, Brockton, 129 West Elm St.
C. D. McCann, Brockton, 12 Cottage St., P. R. C.
R. C. McLeod, Brockton, Goddard Hospital, Sec.
G. A. Moore, Brockton, 167 Newbury St., E. C.
B. H. Peirce, South Hanson, Plymouth County Hospital, M. N. C.
E. L. Perry, Middleboro, 39 Oak St.

SUFFOLK

C. C. Lund, Boston 15, 20 Gloucester St., V. P.
H. L. Albright, Boston 15, 412 Beacon St.
A. W. Allen, Boston 16, 266 Beacon St., C.
J. W. Bartol, Boston, 1 Chestnut St., Ex-Pres.
W. H. Blanchard, Chelsea 50, Soldiers' Home.
W. J. Brickley, Boston 15, 274 Boylston St.
W. E. Browne, Boston 15, 587 Beacon St., Leg. C.
A. M. Butler, Boston 14, Massachusetts General Hospital
A. J. A. Campbell, Boston 15, 520 Commonwealth Ave., E. C.
David Cheever, Boston 16, 193 Marlborough St.
J. F. Conlin, Boston, 121 Park Drive.
Pasquale Costanza, East Boston 28, 238 Maverick St.
N. W. Faxon, Boston 14, Massachusetts General Hospital
Jacob Fine, Boston 15, 330 Brookline Ave.
Reginald Fitz, Boston 15, 319 Longwood Ave., Ex-Pres.
Maurice Fremont-Smith, Boston 15, 12 Hereford St.
Channing Frothingham, Boston, Office Jamaica Plain 30, 1153 Centre St., Ex-Pres.
Joseph Garland, Boston 16, 266 Beacon St.
R. L. Goodale, Boston 16, 330 Dartmouth St., Sec.
A. P. Heusner, Boston 18, 818 Harrison Ave.
John Homans, Boston 16, 311 Beacon St.
A. A. Hornor, Boston 15, 319 Longwood Ave., M. N. C., P. R. C.
L. M. Hurxthal, Boston 15, 605 Commonwealth Ave.
C. S. Keefer, Boston 18, 65 East Newton St.
H. A. Kelly, Winthrop 52, 200 Pleasant St.
T. H. Lanman, Boston 15, 300 Longwood Ave.
R. I. Lee, Boston 16, 264 Beacon St., Ex-Pres.
Donald Munro, Boston 18, 818 Harrison Ave.
R. N. Nye, Boston 15, 8 Fenway
F. R. Ober, Boston 16, 234 Marlborough St., Ex-Pres.
F. W. O'Brien, Boston 15, 465 Beacon St.
J. P. O'Hare, Boston 15, 520 Commonwealth Ave.
L. E. Parkins, Boston 15, 12 Bay State Rd.
L. E. Phaneuf, Boston 16, 270 Commonwealth Ave.
Helen S. Pittman, Boston 16, 264 Beacon St.
J. H. Pratt, Boston 11, 30 Bennet St.
W. H. Robey, Boston 16, 202 Commonwealth Ave., Ex-Pres.
H. F. Root, Boston 15, 81 Bay State Rd., A. E. C.
R. M. Smith, Boston 16, 330 Dartmouth St., C.
M. C. Sosman, Boston 15, 721 Huntington Ave.
Augustus Thorndike, Boston 15, 319 Longwood Ave.
J. J. Todd, Boston 15, 587 Beacon St.
S. N. Vose, Boston 15, 29 Bay State Rd.

Conrad Wesselhoeft, Boston, 315 Marlborough St., A. M. N. C.
C. F. Wilinsky, Boston 15, 330 Brookline Ave.

WORCESTER
L. M. Felton, Worcester, 36 Pleasant St., V. P.
C. R. Abbott, Clinton, 60 Walnut St.
A. W. Atwood, Worcester, 390 Main St.
George Ballantyne, Worcester, 27 Elm St.
F. P. Bousquet, Worcester (8), 390 Main St., A. E. C., A. M. N. C. (interim appointment)
W. P. Bowers, Clinton, 264 Chestnut St., Ex-Pres.
J. J. Cohen, Worcester, 340 Main St.
E. J. Crane, Holden, Armington Lane.
G. R. Dunlop, Worcester, 27 Elm St., Leg. C.
W. J. Elliott, Worcester, 119 Belmont St.
John Fallon, Worcester, 390 Main St.
R. H. Goodale, Worcester, 36 Otsego Rd.
Thomas Hunter, Shrewsbury, 545 Main St.
H. L. Kirkendall, Worcester, 27 Elm St.
J. A. Lundy, Oxford, 26 Main St.
D. K. McClusky, Worcester, 7 Hawthorne St.
J. M. Olson, Westboro, 54 West Main St.
F. A. O'Toole, Clinton, 181 Chestnut St.
R. S. Perkins, Worcester, 27 Elm St., M. N. C.
E. L. Richmond, Worcester (8), 390 Main St. (interim appointment)
N. S. Scarcello, Worcester, 1 Shelden St., P. R. C.
R. F. Sullivan, Worcester, 54 Hillcroft Ave.
J. J. Tegelberg, Worcester, 390 Main St., Sec.
G. C. Tully, Worcester, 1 Cedar St.
R. J. Ward, Worcester, 9 Bellevue St., C.
B. C. Wheeler, Worcester, 27 Elm St., E. C.

WORCESTER NORTH
R. F. Bachmann, Fitchburg, 910 Main St., V. P.
H. C. Arey, Gardner, 66 Parker St.
D. B. Cheetham, Athol, 164 Exchange St.
J. J. Curley, Leominster, 89 West St., A. E. C., M. N. C., Leg. C.
C. B. Gay, Fitchburg, 62 Day St., E. C.
J. V. McHugh, Leominster, 55 West St., A. M. N. C., P. R. C.
J. G. Simmons, Fitchburg, 30 Myrtle Ave., Sec.

The initials *E. C.* following the name of a councilor indicate that he is a member of the Executive Committee, and *A. E. C.* that he is an alternate member of the Executive Committee; *M. N. C.* that he is a member of the Committee on Nominations, and *A. M. N. C.* that he is an alternate member of the Committee on Nominations; *Leg. C.* that he is a member of the Committee on Legislation; *P. R. C.* that he is a member of the Committee on Public Relations; *V. P.* that a member is a councilor by virtue of his office as president of a district society and so vice-president of the general society; *C.* by virtue of his office as chairman of a standing committee; *Sec.* by virtue of his office as secretary of a district society and *Ex-Pres.* by virtue of being a past president.

## CENSORS FOR 1946–1947

BARNSTABLE
(Supervisor to be appointed.)
E. F. Curry, Sagamore.
J. I. B. Vail, Hyannis.
D. H. Hiebert, Provincetown.
Frank Travers, Barnstable.

BERKSHIRE
I. S. F. Dodd, Pittsfield, *supervisor.*
A. C. England, Pittsfield.
W. T. Frawley, Pittsfield.
C. T. Leslie, Pittsfield.
J. W. Bunce, North Adams.

BRISTOL NORTH
J. L. Murphy, Taunton, *supervisor.*
H. G. Vaughan, Attleboro.
A. J. Leddy, Taunton.
L. E. Butler, Taunton.
J. H. Brewster, Attleboro.

BRISTOL SOUTH
Henry Wardle, Fall River, *supervisor.*
E. A. McCarthy, Fall River.
F. M. Howes, New Bedford.
W. F. MacKnight, Fall River.
C. C. Persons, New Bedford.

ESSEX NORTH
R. V. Baketel, Methuen, *supervisor.*
L. C. Peirce, Newburyport.
C. H. Birdsall, Haverhill.
Abraham Ash, Lawrence.
F. R. Radcliffe, Haverhill.

ESSEX SOUTH
A. E. Parkhurst, Beverly, *supervisor.*
S. N. Gardner, Salem.
W. C. Inman, Danvers.
C. F. Twomey, Lynn.
H. N. Baker, Rockport.

FRANKLIN
J. E. Moran, Greenfield, *supervisor.*
W. J. Pelletier, Turners Falls.
J. P. Colleran, South Deerfield.
H. R. Mahar, Orange.
F. W. Dean, Northfield.

HAMPDEN
Frederic Hagler, Springfield, *supervisor.*
J. M. Gilchrist, Springfield.
G. D. Henderson, Holyoke.
John Pallo, Westfield.
J. L. Smead, Springfield.

HAMPSHIRE
L. B. Pond, Easthampton, *supervisor.*
T. F. Corriden, Northampton.
M. E. Cooney, Northampton.
J. E. Hayes, Northampton.
Stephen Brown, Northampton.

MIDDLESEX EAST
M. J. Quinn, Winchester, *supervisor.*
J. H. Fay, Melrose.
S. H. Moses, Winchester.
C. R. Baisley, Reading.
T. P. Devlin, Stoneham.

MIDDLESEX NORTH
W. F. Ryan, Lowell, *supervisor.*
F. R. Brady, Lowell.
R. C. Stewart, Lowell.
H. L. Leland, Lowell.
J. D. Sweeney, Lowell.

MIDDLESEX SOUTH
H. Q. Gallupe, Waltham, *supervisor.*
E. H. Robbins, Somerville.
J. E. Dodd, Framingham.
J. F. Williams, Everett.
H. J. Crumb, Lexington.

NORFOLK
Hyman Morrison, Roxbury, *supervisor.*
C. E. Allard, Dorchester.
H. A. Novack, Brookline.
E. E. O'Neil, Chestnut Hill.
Kathleyne S. Snow, Jamaica Plain.

NORFOLK SOUTH
F. A. Bartlett, Wollaston, *supervisor* (interim appointment).
R. E. Ross, South Braintree.
W. L. Sargent, Quincy.
Arthur Rappeport, Quincy.
H. S. Reid, Cohasset.

PLYMOUTH
E. L. Perry, Middleboro, *supervisor.*
D. W. Pope, Brockton.
R. E. Swenson, Plymouth.
G. A. Buckley, Brockton.
Jacob Brenner, North Easton.

SUFFOLK
J. H. Pratt, Boston, *supervisor.*
H. T. Hutchins, Boston.
A. J. A. Campbell, Boston.
W. E. Browne, Boston.
L. M. Hurxthal, Boston.

WORCESTER
B. C. Wheeler, Worcester, *supervisor.*
J. W. McKoan, Jr., Worcester.
H. K. Spangler, Worcester.
E. J. Crane, Holden.
J. B. Butts, Worcester.

WORCESTER NORTH
C. B. Gay, Fitchburg, *supervisor.*
G. P. Keaveny, Fitchburg.
J. W. Mason, Ashburnham.
E. B. Hopkins, Ayer.
W. E. Currier, Leominster.

## VICE-PRESIDENTS OF THE MASSACHUSETTS MEDICAL SOCIETY (*Ex-Officiis*) FOR 1946–1947

PRESIDENTS OF DISTRICT MEDICAL SOCIETIES

(Arranged according to seniority of fellowship in the Massachusetts Medical Society)

ESSEX SOUTH — Charles A. Worthen, Lynn.
PLYMOUTH — Walter H. Pulsifer, Whitman.
MIDDLESEX SOUTH — John F. Casey, Boston.
MIDDLESEX NORTH — William F. Ryan, Lowell.
NORFOLK SOUTH — David L. Belding, Boston.
SUFFOLK — Charles C. Lund, Boston.
ESSEX NORTH — Guy L. Richardson, Haverhill.
BRISTOL SOUTH — Salmon P. Wilde, New Bedford.
WORCESTER — Lester M. Felton, Worcester.
BERKSHIRE — N. Newall Copeland, Pittsfield.
BRISTOL NORTH — William M. Stobbs, Attleboro.
HAMPDEN — Adolph Franz, Jr., Holyoke.
BARNSTABLE — Harold F. Rowley, Harwich Port.
WORCESTER NORTH — Rudolf F. Bachmann, Fitchburg.
FRANKLIN — John B. Temple, Shelburne Falls.
HAMPSHIRE — John J. Curran, Northampton.
NORFOLK — John H. Cauley, Dorchester.
MIDDLESEX EAST — Walter H. Flanders, Melrose.

## COMMISSIONERS OF TRIAL FOR 1946–1947

BARNSTABLE — F. O. Cass, Provincetown.
BERKSHIRE — J. B. Thomes, Pittsfield.
BRISTOL NORTH — J. W. Cook, Mansfield.
BRISTOL SOUTH — A. C. Lewis, Fall River.
ESSEX NORTH — F. W. Anthony, Haverhill.
ESSEX SOUTH — O. C. Blair, Lynn.
FRANKLIN — K. W. D. Jacobus, Turners Falls.
HAMPDEN — F. K. Dutton, Springfield.
HAMPSHIRE — W. M. Dobson, Northampton.
MIDDLESEX EAST — I. W. Richardson, Wakefield.
MIDDLESEX NORTH — J. F. Boyle, Lowell.
MIDDLESEX SOUTH — H. P. Stevens, Cambridge.
NORFOLK — W. J. Walton, Dorchester.
NORFOLK SOUTH — F. A. Bartlett, Wollaston.
PLYMOUTH — J. A. Carriulo, Brockton.
SUFFOLK — J. R. Torbert, Boston.
WORCESTER — W. P. Bowers, Clinton.
WORCESTER NORTH — H. C. Arey, Gardner.

## OFFICERS OF THE SECTIONS FOR 1946–1947

SECTION OF MEDICINE
*Chairman*, Daniel J. Ellison, Lowell; *vice-chairman*, Francis C. Hall, Boston; *secretary*, Laurence B. Ellis, Boston.

SECTION OF SURGERY
*Chairman*, Alexander J. A. Campbell, Boston; *secretary*, George Reynolds, Pittsfield.
Executive Committee — E. Parker Hayden, Boston; Charles C. Lund, Boston; Edward L. Peirson, Salem.

SECTION OF PEDIATRICS
*President*, Hyman Green, Boston; *secretary*, Gerald N. Hoeffel, Boston and Cambridge.
Executive Committee: *Chairman*, James Marvin Baty, Belmont and Brookline; Leroy T. Stokes, Haverhill; Floyd R. Smith, Pittsfield.

SECTION OF OBSTETRICS AND GYNECOLOGY
*Chairman*, William J. McDonald, Boston; *vice-chairman*, Arthur T. Hertig, Boston; *secretary*, George Van S. Smith, Brookline.

SECTION OF RADIOLOGY
*Chairman*, Edward B. D. Neuhauser, Cambridge and Boston; *secretary*, Hugh F. Hare, Newton and Boston.

SECTION OF PHYSIOTHERAPY
*Chairman*, William D. McFee, Haverhill; *secretary*, Henry A. Tadgell, Belchertown.

SECTION OF DERMATOLOGY AND SYPHILOLOGY
*Chairman*, Jacob Swartz, Brookline; *secretary*, Francis Thurmon, Boston.

SECTION OF ANESTHESIOLOGY
*Chairman*, Sidney C. Wiggin, Waban; *secretary*, Morris J. Nicholson, Boston.

## OFFICERS OF THE DISTRICT MEDICAL SOCIETIES FOR 1946–1947

BARNSTABLE — *President*, Harold F. Rowley, Harwich Port; *vice-president*, Arthur J. D'Elia, Harwich Port; *secretary*, Oscar S. Simpson, Centreville; *treasurer*, Frank Travers, Barnstable; *librarian*, Sheldon L. Hunt, Yarmouth Port; *executive councilor*, Paul M. Butterfield, Harwich; *public-relations councilor*, Paul P. Henson, Hyannis.

BERKSHIRE — *President*, N. Newall Copeland, Pittsfield; *vice-president*, Modestino Criscitiello, Pittsfield; *secretary*, Daniel N. Beers, Pittsfield; *treasurer*, Theodore W. Jones, Pittsfield; *executive councilor*, Isaac S. F. Dodd, Pittsfield; *legislative councilor*, John Hughes, Pittsfield; *public-relations councilor*, Patrick J. Sullivan, Dalton.

BRISTOL NORTH — *President*, William M. Stobbs, Attleboro; *vice-president*, Joseph V. Chatigny, Taunton; *secretary*, William J. Morse, Attleboro; *treasurer*, Charles E. Hoye, Taunton; *executive councilor*, Joseph L. Murphy, Taunton; *legislative councilor*, Curtis B. Kingsbury, Taunton; *public-relations councilor*, Milton E. Johnson, Attleboro.

BRISTOL SOUTH — *President*, S. Perry Wilde, New Bedford; *vice-president*, George W. Blood, Fall River; *secretary and treasurer*, James E. Fell, Fall River; *executive councilor*, Richard B. Butler, Fall River; *legislative councilor*, Curtis C. Tripp, New Bedford; *public-relations councilor*, Harold E. Perry, New Bedford.

ESSEX NORTH — *President*, Guy L. Richardson, Haverhill; *vice-president*, Lincoln C. Peirce, Newburyport; *secretary*, Harold R. Kurth, Methuen; *treasurer*, J. LeRoy Wood, Methuen; *executive councilor*, Rolf C. Norris, Methuen; *legislative councilor*, Nicandro F. DeCesare, Methuen; *public-relations councilor*, Harold R. Kurth, Methuen.

ESSEX SOUTH — *President*, Charles A. Worthen, Lynn; *vice-president*, Hubert A. Boyle, Middleton; *secretary*, Henry D. Stebbins, Marblehead; *treasurer*, Andrew Nichols, 3d, Danvers; *executive councilor*, Walter G. Phippen, Salem; *public-relations councilor*, Loring Grimes, Swampscott.

FRANKLIN — *President*, John B. Temple, Shelburne Falls; *vice-president*, Lawrence R. Dame, Greenfield; *secretary and treasurer*, Harry L. Craft, Ashfield; *executive councilor*, John E. Moran, Greenfield; *legislative councilor*, Arthur W. Hayes, Greenfield; *public-relations councilor*, Frank A. Millett, Greenfield.

HAMPDEN — *President*, Adolph Franz, Jr., Holyoke; *vice-president*, Arthur F. G. Edgelow, Springfield; *secretary and treasurer*, George C. Steele, West Springfield; *executive councilor*, William A. R. Chapin, Springfield; *legislative councilor*, Arthur H. Riordan, Indian Orchard; *public-relations councilor*, Patrick E. Gear, Holyoke.

HAMPSHIRE — *President*, John J. Curran, Northampton; *vice-president*, Edward J. Manwell, Northampton; *secretary and treasurer*, Mary P. Snook, Worthington; *librarian*, Abbie M. O'Keefe, Northampton; *executive councilor*, Henry A. Tadgell, Belchertown; *legislative councilor*, Lawrence N. Durgin, Amherst; *public-relations councilor*, Joseph R. Hobbs, Williamsburg.

MIDDLESEX EAST — *President*, Walter H. Flanders, Melrose; *vice-president*, Daniel L. Joyce, Woburn; *secretary*, Roy W. Layton, Melrose; *treasurer*, Albert E. Small, Melrose; *librarian*, Angelo L. Maietta, Winchester; *executive councilor*, Kenneth L. Maclachlan, Melrose; *legislative councilor*, John M. Wilcox, Woburn; *public-relations councilor*, Milton J. Quinn, Winchester.

MIDDLESEX NORTH — *President*, William F. Ryan, Lowell; *vice-president*, C. Stoyle Baker, Lowell; *secretary*, Brendan D. Leahey, Lowell; *treasurer*, Mason D. Bryant, Lowell; *executive councilor*, William F. Ryan, Lowell; *legislative councilor*, Archibald R. Gardner, Lowell; *public-relations councilor*, Daniel J. Ellison, Lowell.

MIDDLESEX SOUTH — *President*, John F. Casey, Allston; *vice-president*, Arthur M. Jackson, Everett; *secretary*, Alexander A. Levi, Newton Centre; *treasurer*, Fabyan Packard, Belmont; *orator*, Harold E. MacMahon, Cambridge; *executive councilor*, Harold G. Giddings, Newton Centre; *legislative councilor*, Kenneth J. Tillotson, Belmont; *public-relations councilor*, Gordon M. Morrison, Waban.

NORFOLK — *President*, John H. Cauley, Dorchester; *vice-president*, Carl Bearse, Newton; *secretary*, Basil E. Barton, West Roxbury; *treasurer*, Frederick Reis, Jamaica Plain; *executive councilor*, Charles J. Kickham, Brookline; *public-relations councilor*, Dean S. Luce, Canton.

NORFOLK SOUTH — *President*, David L. Belding, Hingham; *vice-president*, Robert L. Cook, Quincy; *secretary and librarian*, Ebenezer K. Jenkins, Braintree; *treasurer*, Francis G. King, North Quincy; *executive councilor*, Daniel B. Reardon, Quincy; *legislative councilor*, David L. Belding, Hingham; *public-relations councilor*, Henry A. Robinson, Hingham.

PLYMOUTH — *President*, Walter H. Pulsider, Whitman; *vice-president*, Frank E. Wheatley, Brockton; *secretary*, Ralph C. McLeod, Brockton; *treasurer*, Alton L. Hurlburt, East Bridgewater; *librarian*, John H. Weller, State Farm; *executive councilor*, George A. Moore, Brockton; *legislative councilor*, Alfred L. Duncombe, Brockton; *public-relations councilor*, Charles D. McCann, Brockton.

SUFFOLK — *President*, Charles C. Lund, Boston; *vice-president*, Hollis L. Albright, Boston; *secretary*, Robert L. Goodale, Boston; *treasurer*, Richard S. Eustis, Boston; *executive councilor*, Alexander J. A. Campbell, Boston; *legislative councilor*, William E. Browne, Boston; *public-relations councilor*, Albert A. Hornor, Boston.

WORCESTER — *President*, Lester M. Felton, Worcester; *vice-president*, Frank B. Carr, Worcester; *secretary*, Julius J. Tegelberg, Worcester; *treasurer*, Edward P. Disbrow, Worcester; *librarian*, John Fallon, Worcester; *executive councilor*, Bancroft C. Wheeler, Worcester; *legislative councilor*, George R. Dunlop, Worcester; *public-relations councilor*, Nicholas S. Scarcello, Worcester.

WORCESTER NORTH — *President*, Rudolf F. Bachmann, Fitchburg; *vice-president*, Francis A. Reynolds, Athol; *secretary*, James G. Simmons, Fitchburg; *treasurer*, Frederick H. Thompson, Jr., Fitchburg; *executive councilor*, C. Bertram Gay, Fitchburg; *legislative councilor*, John J. Curley, Leominster; *public-relations councilor*, James V. McHugh, Leominster.

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## ADMISSIONS RECORDED FROM MAY 23, 1945, TO MAY 22, 1946

| YEAR OF ADMISSION | NAME AND RESIDENCE | MEDICAL SCHOOL |
|---|---|---|
| 1946 | *Abrams, Sidney Martin, Malden | Middlesex |
| 1946 | Abodeely, Robert Assad, Worcester | Boston University |
| 1946 | Achin, Joffre Victor, Charlton City | Tufts |
| 1946 | Allen, Roger Everett, Shrewsbury | Tufts |
| 1946 | *Andaloro, Vincent A., Everett | Middlesex |
| 1946 | Anderson, Fred A., West Roxbury | Boston University |
| 1945 | Anderson, John Clifford, Stoneham | Tufts |
| 1945 | Angelo, Peter, Waltham | Tufts |
| 1945 | Antonellis, Carl Joseph, Cambridge | Tufts |
| 1946 | Appleford, George Burton, Springfield | Tufts |
| 1946 | Apshaga, Bronie John Peter, Brighton | Tufts |
| 1945 | *Arakelian, Michael John, Methuen | Kansas City University of Physicians and Surgeons |
| 1945 | Arbeene, George Norman, Somerville | Tufts |
| 1945 | Ascher, David Samuel, Brighton | Tufts |
| 1945 | Astwood, Edwin Bennett, Brookline | McGill University |
| 1946 | *Atwood, Eldridge Delories, Woburn | College of Physicians and Surgeons, Boston |
| 1946 | Austen, George, Jr., Brookline | Harvard |
| 1946 | Awramik, Stanley Maryan, Lynn | Boston University |
| 1945 | Bacon, William Benjamin, Brookline | Harvard |
| 1946 | Baker, William Jessamin, Cambridge | Harvard |
| 1946 | Balboni, Victor G., Boston | Harvard |
| 1945 | *Bannerman, Donald Bruce, East Bridgewater | Middlesex |
| 1946 | *Barbieri, Joseph Gene, Lawrence | Middlesex |
| 1945 | *Barrette, Rene A., Fall River | College of Physicians and Surgeons, Boston |
| 1945 | Bellinger, Martin John, Boston | Harvard |
| 1946 | Bengloff, Harold, Brookline | Harvard |
| 1946 | Bennett, Robert Edward, Worcester | Tufts |
| 1946 | *Berenson, William, Lynn | Middlesex |
| 1946 | Berg, Milton Louis, Everett | Harvard |
| 1946 | Berry, Francis Leonard, Milford | Tufts |
| 1945 | Bilodeau, Charles Cloudman, Longmeadow | Columbia University |
| 1946 | Binder, Eugene Norman, Chestnut Hill | Boston University |
| 1946 | Blanchard, Herbert Norman, Hingham | Boston University |
| 1946 | *Blank, Joseph George, Waltham | Middlesex |
| 1946 | *Blesoff, Benjamin, Somerville | Middlesex |
| 1945 | Block, Herman Leonard, Roxbury | Boston University |
| 1946 | Blodgett, James Thomas, West Boylston | Harvard |
| 1945 | Bloombergh, John Hollis, Chestnut Hill | Tufts |
| 1946 | Bluhm, Samuel, Dorchester | Tufts |
| 1946 | Bolduc, Robert Aime, Worcester | Tufts |
| 1945 | Botsford, Thomas Winston, Boston | Harvard |

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|---|---|---|
| 1946 | Bourgeois, George Albert, Boston | Tufts |
| 1945 | Braconier, Harry Erland, Belmont | Tufts |
| 1946 | Bragan, James A., Swampscott | Tufts |
| 1946 | Brown, Albert Abraham, Lynn | Boston University |
| 1945 | Brown, Ralph E., Jr., Waltham | Tufts |
| 1946 | Bryer, James Allen, Jr., North Attleboro | Tufts |
| 1945 | *Burian, Hermann Martin, Newton Highlands | University of Belgrade |
| 1945 | Bush, Cecelia Agnes O'Farrell, Brighton | Tufts |
| 1946 | *Campbell, Donald E., Stockbridge | Middlesex |
| 1946 | Capodieci, Florindo Horace, Dorchester | Boston University |
| 1946 | Carleton, William Talbot, Worcester | Harvard |
| 1946 | Cartwright, John Thompson, Randolph | University of Pennsylvania |
| 1946 | Cass, Victoria Maxwell, Winchester | Tufts |
| 1946 | *Castleton, Herbert Edward, Rockland | Middlesex |
| 1945 | *Cerrato, Calvin Michele, Cambridge | University of Bologna |
| 1946 | *Cinella, John Thomas, Lee | Middlesex |
| 1945 | Clark, Glenmore Ford, North Andover | Medico-Chirurgical College (Philadelphia, Pa.) |
| 1946 | Clough, Joseph Messer, Boston | Jefferson Medical College |
| 1945 | Cloyes, Lorraine, West Newbury | Boston University |
| 1946 | Cohen, Manley B., Dorchester | Laval University |
| 1946 | Compton, David Wesley, Boston | University of Pennsylvania |
| 1945 | Connors, John Phillip, Worcester | Georgetown University |
| 1946 | *Cotter, John Thomas, North Attleboro | College of Physicians and Surgeons, Boston |
| 1946 | Cotter, Joseph Robert, Newton Upper Falls | Tufts |
| 1946 | *Covner, Philip David, Lynn | Chicago Medical School |
| 1945 | Cozza, Lawrence Francis, Medford | Tufts |
| 1945 | Crane, Timothy Francis, Cambridge | Columbia University |
| 1946 | Creeden, Francis Vincent, Brockton | Georgetown University |
| 1946 | Croskery, William Francis, Worcester | Boston University |
| 1946 | Currens, James H., Brookline | Duke University |
| 1946 | Curtis, Charles Nason, Salem | Tufts |
| 1946 | Curtis, Sprague, Springfield | Tufts |
| 1946 | *Davis, Albert, Quincy | Middlesex |
| 1945 | *DeCesare, Frank J., Methuen | Middlesex |
| 1945 | *DeFeo, Amadeo Joseph, Everett | Middlesex |
| 1946 | DeMinico, Luigi B., Jamaica Plain | Tufts |
| 1946 | Derrick, Charles Campbell, Springfield | University of Chicago |
| 1945 | Dorsey, Joseph Farrell, Boston | University of Tennessee |
| 1945 | Duston, Charles H., Boston | Tufts |
| 1946 | Dyer, Edward Carlton, Brookline | Harvard |
| 1946 | Egnatz, Nicholas, Jr., Westfield | Indiana University |
| 1946 | Ellis, Daniel Sumner, Cambridge | Harvard |
| 1946 | Elton, Norman William, Newton Highlands | Boston University |
| 1945 | Espinoza, David Valencia, Boston | Tufts |
| 1946 | Eyres, Alfred Ernest, Worcester | Iowa State University |
| 1945 | Fagan, Frederick John, Taunton | Boston University |
| 1945 | Fagan, John Francis, Jr., Dorchester | George Washington University |
| 1945 | Failla, Samuel Di Grandi, Greenfield | University of Georgia |
| 1946 | Famiglietti, Joseph Angelo, East Boston | Tufts |
| 1945 | Ferrarone, Edward Joseph, Springfield | Georgetown University |
| 1946 | Finnell, Ambrose Francis, New Bedford | Georgetown University |
| 1946 | Fiore, Autino, Cambridge | Tufts |
| 1945 | *Fischer-Galati, Theodore Isidore Max, Andover | University of Vienna |
| 1946 | Fisher, Robert Martin, Waltham | Columbia University |
| 1945 | Fitzgerald, Edward Francis, Quincy | Georgetown University |
| 1945 | Fitzpatrick, Audrey Jane, Worcester | Tufts |
| 1945 | Flaschner, Ira, Newton | Boston University |
| 1945 | Ford, Frederick Francis, Waltham | Georgetown University |
| 1946 | *Friberg, Robert Adolph, Winchester | Middlesex |
| 1946 | *Friedland, Fritz, Brookline | University of Berlin |
| 1946 | *Frisch, Joseph, Vineyard Haven | Middlesex |
| 1946 | *Frogel, Reuben Harry, East Braintree | Middlesex |
| 1946 | *Galbo, Samuel Joseph, Shelburne Falls | Middlesex |
| 1945 | Ganem, Emil Joseph, Methuen | Johns Hopkins University |
| 1946 | *Gaspar, Shari Band, Baldwinsville | Royal Hungarian University of Pecs |
| 1946 | Gilmore, Raymond, Lowell | Tufts |
| 1946 | Ginsburg, Edward Merrill, Quincy | Emory University |
| 1945 | Giuliano, Charles Edward, Brookline | Baylor University |
| 1946 | *Gold, Abraham, Chelsea | Middlesex |
| 1945 | *Goldblatt, Harold William, Lawrence | Medical School, Royal Colleges |
| 1946 | Golden, Theodore Solomon, Newton | Tufts |
| 1946 | *Goldman, Herbert, Lynn | Middlesex |
| 1946 | Goodale, Walter Temple, Weston | Harvard |
| 1945 | Gorfine, Robert, Allston | St. Louis University |
| 1945 | Gould, Louis Nathan, Northampton | New York Medical College |
| 1946 | *Green, Max Benjamin, Lawrence | Middlesex |
| 1945 | Greenblatt, Milton, Boston | Tufts |
| 1945 | Gregory, Elizabeth A., Boston | Boston University |
| 1946 | Grendal, Michael Francis, Roslindale | Jefferson Medical College |
| 1946 | Guralnick, Eugene, Roxbury | Tufts |
| 1946 | Haase, Ferdinand, Jr., Boston | Harvard |
| 1945 | Haley, William Thomas, Jr., Boston | Tufts |

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|---|---|---|
| 1945 | Halpin, William Joseph, Medford | Tufts |
| 1946 | *Hamburger, Herbert, Worcester | University of Pisa |
| 1946 | Hamlin, Hannibal, Weston | Yale |
| 1946 | *Hannon, Harland N., North Grafton | Kansas City University of Physicians and Surgeons |
| 1945 | Hardy, Irad Benjamin, Jr., Waltham | Harvard |
| 1945 | *Hauck, Charles Wilfred, Brookline | Middlesex |
| 1946 | Heath, Wilmer Porter, Needham | Boston University |
| 1946 | Heeks, William Garland, North Adams | Harvard |
| 1946 | Hogan, Edward Norbert, Dorchester | Georgetown University |
| 1945 | Hogan, Gerald Francis, Amherst | Tufts |
| 1946 | Hunt, Richard Joseph, Stoneham | Tufts |
| 1946 | *Hyfer, Harry Jack, Winthrop | Middlesex |
| 1946 | Isenstein, Charles, Brookline | Tufts |
| 1945 | *Jacobs, Ezra Astor, Brookline | College of Physicians and Surgeons, Boston |
| 1946 | *Jellinek, Kurt, Middleboro | University of Vienna |
| 1946 | Joel, Samuel W., Belmont | Tufts |
| 1946 | Johnson, Elwood Gordon, Waltham | Tufts |
| 1946 | Johnson, Goodwin Adolph, Brookline | Tufts |
| 1946 | Kane, Francis Clement, Salem | Tufts |
| 1946 | *Karp, Isadore Albert, Chelsea | Middlesex |
| 1946 | *Karp, Lewis S., Worcester | Middlesex |
| 1946 | Kaufman, Sumner, Chelsea | Boston University |
| 1946 | Kaufmann, Gustav Grosvenor, Andover | Rush Medical College |
| 1946 | Keenan, George Francis, West Roxbury | Tufts |
| 1946 | Kibbe, Milton Homer, West Springfield | Tufts |
| 1946 | Kirsh, David, Boston | University of Pennsylvania |
| 1946 | Klein, Leonard Milton, Roxbury | Tufts |
| 1946 | Knox, Barron D., Springfield | Tufts |
| 1945 | Koenig, Emil John, Jr., Holden | Tufts |
| 1945 | *Kraus, Paul Stefan, North Grafton | University of Bologna |
| 1946 | *Kretzmer, Eugene, Worcester | University of Munich |
| 1945 | Lamphier, Timothy Andre, Wellesley Hills | Boston University |
| 1946 | Landsteiner, Ernest Karl, Newton Centre | Harvard |
| 1945 | Lapidus, Bernard, Dorchester | Tufts |
| 1946 | Leary, Howard Leo, Jamaica Plain | Tufts |
| 1945 | Leonard, John Charles, Melrose | Yale |
| 1946 | Levenson, Stanley Mortimer, Boston | Harvard |
| 1946 | Levine, Harold David, Brookline | Harvard |
| 1946 | *Levine, Louis, New Bedford | Middlesex |
| 1946 | Levine, Saul Charles, Malden | Tufts |
| 1946 | Lewis, Donald King, Winchester | University of Michigan |
| 1945 | Linenthal, Arthur Joseph, Brookline | Harvard |
| 1946 | *Litton, Nina, Brookline | University of Naples |
| 1945 | Livingston, Stanton Knowlton, West Roxbury | University of Virginia |
| 1946 | *London, Clarence, Lynn | Middlesex |
| 1945 | Mahaney, William F., Norwood | University of Pennsylvania |
| 1946 | *Mandrachia, Alfonso Charles, Everett | Middlesex |
| 1946 | Martinson, Melvin Stanley, Acton | Tufts |
| 1946 | Matson, Donald Darrow, Boston | Harvard |
| 1945 | McAuliffe, Eugene Francis, Jr., Boston | Georgetown University |
| 1945 | McCann, John Buckley, Brockton | Georgetown University |
| 1946 | *McCarthy, Allan Joseph, Arlington | Middlesex |
| 1946 | McCarthy, Thomas Francis, Lowell | Harvard |
| 1945 | McCormack, George Augustine, Medford | Hahnemann Medical College |
| 1946 | McDonough, Francis Edward, West Newton | University of Wisconsin |
| 1945 | McDonough, James Francis, Woburn | Tufts |
| 1946 | *McKerr, Frederick Gunby, Pittsfield | Chicago Medical School |
| 1946 | Mead, Sedgwick, Littleton | Harvard |
| 1946 | Michaud, Gerard Norman, Salem | Tufts |
| 1945 | *Migliore, Joseph O., Malden | Middlesex |
| 1946 | *Mills, Arnold, Mattapan | Middlesex |
| 1946 | Milner, Leo Robert, Brookline | Tufts |
| 1946 | Miner, Theodore Richardson, Longmeadow | New York University |
| 1946 | Moran, Richard James, Arlington | Tufts |
| 1945 | Mousselet, Mabel Weaver, Boston | Boston University |
| 1946 | Mulcahy, Richard Edward, Uxbridge | Tufts |
| 1945 | *Murphy, John Patrick, Jamaica Plain | Mid-West Medical College |
| 1946 | Murray, Martin B., Springfield | New York University |
| 1945 | Nadas, Alexander S., Greenfield | Wayne University |
| 1946 | Nash, William Cornelius, Lynn | St. Louis University |
| 1945 | Neber, Jacob, Newton Centre | Tufts |
| 1945 | *Nichols, Charles Irving, Medford | Middlesex |
| 1946 | *Noonan, John Philip, Brockton | Middlesex |
| 1945 | *Nussbaum, Julius, Lynn | University of Vienna |
| 1946 | Oliver, Howard Murray, Worcester | Jefferson Medical College |
| 1946 | *Olson, John Herbert, Colrain | College of Physicians and Surgeons, Boston |
| 1946 | Palmer, Edward Joseph, Lexington | Harvard |
| 1946 | *Parton, George Povey, Jr., Wellesley | Middlesex |
| 1945 | Peabody, Stephen Davis, Newburyport | Tufts |
| 1946 | Pearson, Robert Winsor, Beverly | Harvard |
| 1946 | Pekala, Stanley John, Pittsfield | University of Vermont |
| 1946 | *Pinzino, Charles James, Brockton | Kansas City University of Physicians and Surgeons |

| | | |
|---|---|---|
| 1945 | Pollen, Abraham, Chelsea | Tufts |
| 1946 | Portman, Harry, Malden | Boston University |
| 1946 | Powell, Thurston Gates, Auburndale | Jefferson Medical College |
| 1946 | Pratt, Thomas Dennie, Andover | Yale |
| 1946 | *Press, Harry Alvin, Pittsfield | Middlesex |
| 1946 | *Prochnik, James Jonas, Allston | University of Vienna |
| 1946 | Rawson, Rulon Wells, Cambridge | Northwestern University |
| 1946 | Reardon, Edward Richard, Worcester | Tufts |
| 1945 | *Receputo, Paul, Somerville | Middlesex |
| 1946 | Regan, Francis Clinton, Wellesley | University of Rochester |
| 1945 | *Reinisch, Max, Holyoke | University of Prague |
| 1945 | *Reyersbach, Gertrud Clara, Boston | University of Gottingen |
| 1946 | Richer, Arthur George, Hudson | Tufts |
| 1945 | Rini, Joseph, Springfield | Long Island College of Medicine |
| 1945 | Rodofsky, Milton Harold, Dorchester | Tufts |
| 1946 | Rogell, David, Allston | Tufts |
| 1946 | Rogers, Daniel Miner, South Hamilton | Harvard |
| 1945 | Roseman, Melvin David, Dorchester | Boston University |
| 1946 | *Rosenbaum, Emil Elias, Lowell | University of Berlin |
| 1945 | Rosenfeld, Leon, Brookline | Harvard |
| 1946 | Ross, Frederick Phelps, Brookline | Harvard |
| 1945 | Ross, J. Newton, Brookline | University of Illinois |
| 1945 | Ross, John Richard, Needham | Ohio State University |
| 1946 | *Rothschild, Alfred F., Worcester | University of Munich |
| 1945 | Russo, Edward Attilio, Boston | Tufts |
| 1946 | Ryack, Leon, Brookline | New York Medical College |
| 1946 | *Saffran, Irving, Mattapan | Middlesex |
| 1945 | Sall, Robert Dayton, Mattapan | Harvard |
| 1945 | Salwen, Robert, Brookline | Boston University |
| 1946 | Scanlon, James Gerald, Worcester | Hahnemann Medical College |
| 1946 | *Schechtman, Harold I., Fitchburg | Middlesex |
| 1945 | Schier, Woodrow Wilson, Boston | Columbia University |
| 1946 | *Schmoyer, Sheron Aaron Alfred, Pittsfield | Middlesex |
| 1946 | Schofield, Alan Leslie, Lowell | Tufts |
| 1945 | *Schwartz, Harry H., Everett | Middlesex |
| 1946 | Schwartz, Isadore, Quincy | Tufts |
| 1946 | *Schwartz, Robert, Hyde Park | Middlesex |
| 1946 | Sears, Warren Hooper, Springfield | Johns Hopkins University |
| 1945 | Secunda, Lazarus, Pittsfield | Columbia University |
| 1945 | *Seltzer, Joseph, Newton Centre | Middlesex |
| 1946 | Shands, Harley Cecil, Boston | Tulane University |
| 1946 | Shapiro, Robert, Brookline | Tufts |
| 1946 | Shaughnessy, William Joseph, Worcester | Hahnemann Medical College |
| 1946 | Shaw, Lister Harvey, Lowell | Harvard |
| 1946 | Shea, Charles Michael, Medford | St. Louis University |
| 1946 | Sheddan, Frank G., Jr., Wellesley | Baylor University |
| 1946 | *Short, Leonard V., Lexington | Middlesex |
| 1946 | Silverman, Irving, Cambridge | Boston University |
| 1945 | Simon, Naif Louis, Quincy | Boston University |
| 1945 | Sinish, Kenneth William, Melrose | Harvard |
| 1945 | Smith, Leo Joseph, Dorchester | Tufts |
| 1946 | *Sneiderman, Robert, Lynn | Middlesex |
| 1946 | Snyder, Philip Frank, Lynn | Tufts |
| 1946 | Sommer, Francis Xavier, Newton Centre | Yale |
| 1946 | Sorkin, Joseph James, Dorchester | Chicago College of Medicine and Surgery |
| 1946 | Spadea, Saveria, Brockton | Loyola University |
| 1945 | Squire, Lucy Frank, Boston | Woman's Medical College |
| 1945 | *Staffier, Anthony Ralph, West Somerville | Middlesex |
| 1946 | Starbuck, George William, Fairhaven | University of Vermont |
| 1946 | Stein, Tobias, Boston | Columbia University |
| 1945 | Stewart, Donald Mitchell, Malden | Tufts |
| 1946 | *Strassmann, George Samuel, Waltham | University of Heidelberg |
| 1946 | Stratton, Charles William, Lee | Albany Medical College |
| 1946 | Sturdevant, Charles Lyon, Newtonville | University of Nebraska |
| 1946 | Sturnick, Melvin Irving, Allston | Tufts |
| 1946 | Suzedell, Eugene, Quincy | Boston University |
| 1946 | *Sweeney, Guy Raymond, Medford | Middlesex |
| 1945 | Swenson, Orvar, Brookline | Harvard |
| 1945 | *Tauber, Max, Waltham | Middlesex |
| 1946 | *Tedeschi, Pasquale R., Newton | Middlesex |
| 1946 | *Thornton, Patrick John Sinclair, Brookline | University of Lausanne |
| 1946 | *Turanyi, Bela, Dorchester | Royal Hungarian Elisabeth University |
| 1946 | Twigg, Edward James, Winthrop | Tufts |
| 1946 | Vanderlaan, Willard Parker, Jr., Marblehead Neck | Harvard |
| 1946 | Varnum, Joseph Bradley, Lowell | Tufts |
| 1946 | Vassos, George Arthur, Jr., Springfield | Cornell University |
| 1946 | Verde, Aldo Guido, Dorchester | Tufts |
| 1945 | Walker, George Marshall, Jamaica Plain | Cornell University |
| 1945 | *Wantman, Bernard, Somerville | Middlesex |
| 1946 | Ware, Paul Francis, Chestnut Hill | Harvard |
| 1945 | Warren, George Joseph, Waltham | Tufts |
| 1946 | Warren, Joseph Edward, Worcester | Harvard |

| | | |
|---|---|---|
| 1946 | Waskow, Eleanor A., Boston | University of Wisconsin |
| 1946 | Weinstein, Louis, Brighton | Boston University |
| 1946 | Weisman, Avery Danto, Boston | University of Michigan |
| 1946 | Weiss, William Anthony, Boston | Jefferson Medical College |
| 1945 | Welcker, Merrill Louis, Jr., South Duxbury | Tufts |
| 1946 | Westermeyer, Marion Wallace, Melrose | College of Medical Evangelists |
| 1945 | *White, Edward G., Springfield | Chicago Medical School |
| 1945 | Wildberger, William C., West Newton | Boston University |
| 1946 | Wilson, Walter E., Jr., Brookline | Emory University |
| 1946 | Winograd, Abbott Louis, Lynn | University of Michigan |
| 1946 | Wise, Charles Samuel, Cambridge | New York University |
| 1946 | Wood, William Baxter, West Roxbury | Tufts |
| 1946 | Worthen, Charles Arthur, Jr., Lynn | Tufts |
| 1946 | Zeltzerman, Israel, Roxbury | Tufts |
| 1946 | Zupanec, Ralph, Pittsfield | University of Kansas |

Total.....302

*The candidate, after a personal interview, was approved by the Committee on Membership and permitted to take an examination before a board of censors.

## Deaths Reported from May 23, 1945, to May 22, 1946

| ADMITTED | NAME | PLACE OF DEATH | DATE OF DEATH | AGE |
|---|---|---|---|---|
| 1931 | Alexandrov, Vitaly John | Rutland | March 5, 1946 | 55 |
| 1933 | Anshin, Marcus Moses | Lynn | May 21, 1945 | 52 |
| 1905 | Atwater, James Billings | Westfield | May 3, 1946 | 87 |
| 1903 | Bartley, John Joseph | Lawrence | June 16, 1945 | 73 |
| 1884 | †Baxter, Edward Hooker | Hyde Park | July 30, 1945 | 93 |
| 1914 | Bresnihan, Frank Nesdel | Cambridge | July 29, 1945 | 58 |
| 1921 | †Brodrick, Francis Sidney | Boston | September 11, 1945 | 77 |
| 1917 1936 | Buck, William Edgar | Randolph | September 15, 1945 | 58 |
| 1906 | Burgess, Charles James | Lawrence | February 16, 1946 | 76 |
| 1898 | Cabot, Hugh | Ellsworth, Maine | August 14, 1945 | 73 |
| 1904 | Cannon, Walter Bradford | Franklin, New Hampshire | October 1, 1945 | 73 |
| 1925 | Cavanaugh, Mortimer Thomas | Great Barrington | October 7, 1945 | 73 |
| 1890 1919 | †Churchill, Frank Spooner | Bass River | February 27, 1946 | 81 |
| 1892 | †Cousens, Nicholas William | Waltham | March 19, 1946 | 81 |
| 1914 1938 | Cox, Oscar Francis | Brookline | June 3, 1945 | 57 |
| 1905 1943 | Cregg, Francis Aloysius | Methuen | November 30, 1945 | 64 |
| 1932 | Davenport, Benita Coolidge | Middleton | December 20, 1945 | 57 |
| 1905 | Davison, Arthur Howard | Milton | September 26, 1945 | 71 |
| 1905 | †Dexter, Fred Fay | Springfield | July 10, 1945 | 66 |
| 1898 | †Dickson, Richard Ensign | Holyoke | May 8, 1945 | 77 |
| 1888 | †Drummey, Nicholas Daniel | Dorchester | December 18, 1945 | 80 |
| 1913 | †Edsall, David Linn | Tryon, North Carolina | August 12, 1945 | 76 |
| 1927 | Elliott, John Joseph | West Roxbury | March 29, 1946 | 49 |
| 1904 | Evans, Miner Harlow Amos | Wellesley Hills | May 5, 1946 | 65 |
| 1919 | †Fessenden, Charles Hill | Newton Centre | April 19, 1946 | 82 |
| 1892 | †Goodwin, James Joseph | Clinton | December 29, 1945 | 80 |
| 1892 1923 | Goray, James Philip | Fitchburg | November 12, 1945 | 78 |
| 1929 | Greenway, Major Thomas Herbert, M.C., A.U.S. | Framingham | June 23, 1945 | 44 |
| 1896 | †Griswold, Merton Lyman | Uxbridge | June 10, 1945 | 76 |
| 1914 | Gunter, Fred Clarke | Belmont | April 24, 1946 | 54 |
| 1899 | Hammond, William John | East Walpole | July 4, 1945 | 76 |
| 1915 | Harkins, William Joseph | Quincy | August 25, 1945 | 60 |
| 1936 | Hayes, Captain Paul Thomas, M.C., A.U.S. | France | October 1, 1945 | 36 |
| 1915 | Hearn, Walter Lawrence | Lynn | September 30, 1945 | 67 |
| 1942 | Hurlbut, Lt. Robert Satterlee (MC), U.S.N.R. | "U.S.S. Halligan off Okinawa" | March 26, 1945 | 33 |
| 1901 | Jack, Lewis Harlow | West Newton | May 3, 1946 | 73 |
| 1937 | Jacobson, Nathan Louis | Lynn | July 13, 1945 | 60 |
| 1930 | Kaplan, Boris | New Bedford | December 25, 1945 | 56 |
| 1935 | Keith, Theodore Kent | Newton Centre (on terminal leave, A.U.S.) | January 15, 1946 | 43 |
| 1888 | †Lawler, William Patrick | Lowell | December 8, 1945 | 85 |
| 1927 | Lee, Frank Robert | North Andover | October 15, 1945 | 42 |
| 1916 | Leonard, Edward DeWitt | Newton Centre | December 15, 1945 | 60 |
| 1893 | †Libby, Jesse Herbert | Weymouth | September 25, 1945 | 78 |
| 1907 | †Lorimer, Felix | Pomona, California | February 23, 1944 | 79 |
| 1907 1913 1915 | MacLeod, John Malcolm | Quincy | October 19, 1945 | 72 |
| 1905 | †Mayberry, Frank Eugene | Rockland | October 1, 1945 | 85 |
| 1904 | McCausland, William James | Quincy | November 5, 1945 | 70 |
| 1921 1942 | McLeod, Melvin Saunders | Melrose | February 26, 1946 | 49 |

| | | | |
|---|---|---|---|
| 1903 | McNamara, John James | Brockton | January 3, 1946....74 |
| 1904 | †McPherson, George Edwin | Amherst | June 16, 1945....68 |
| 1914 | Metcalf, Richard | Winthrop | May 21, 1945....57 |
| 1912 | Metzger, Butler | Lynn | April 25, 1946....72 |
| 1915 | Moore, George Andrew | Palmer | October 6, 1945....74 |
| 1921 | Morrison, William Henry | Springfield | November 22, 1945....65 |
| 1942 | Mulligan, Francis Joseph | Newton | February 3, 1946....44 |
| 1888 | †Palmer, Sarah Eilen | Boston | August 23, 1945 Unknown |
| 1899 | †Parker, Ralph Walter | Pelham, New Hampshire | February 18, 1944....67 |
| 1904 | †Patterson, Alice Maria | Marblehead | June 20, 1945....75 |
| 1925 | Piper, Comdr. Frank James (MC), U.S.N.R | Framingham (on terminal leave) | April 4, 1946....48 |
| 1903 | Poirier, Horace | North Conway, New Hampshire | August 14, 1945....67 |
| 1896 | †Reddy, Joseph Warren | South Boston | About August 1, 1945..74 |
| 1928 | Richardson, Carl Eugene | Franklin | July 30, 1945....61 |
| 1891 | †Robbins, Fred Gibson | Salem | May 16, 1945....74 |
| 1911 | †Rosenau, Milton Joseph | Chapel Hill, North Carolina | April 9, 1946....77 |
| 1913 | Saunders, Edmund Louis | Wilbraham | May 11, 1944....68 |
| 1933 | Scenna, Donato Theodore | Melrose (on terminal leave, A.U.S.) | March 3, 1946....41 |
| 1924<br>1938 | Silbert, Harry | Salem | October 31, 1945....50 |
| 1891<br>1926 | †Stevens, Harry Lawrence | New Bedford | January 29, 1946....75 |
| 1925 | Stratton, Charles William | Lee | November 6, 1945....69 |
| 1906 | †Sullivan, Cornelius Augustine | Braintree | March 19, 1946....66 |
| 1928 | Talty, Francis Eugene | Hoboken, New Jersey | May 27, 1945....65 |
| 1911 | Timmins, Edward Francis | South Boston | December 11, 1945....64 |
| 1894 | †Treanor, John Peter | Dorchester | May 4, 1946....81 |
| 1903 | Truesdale, Philemon Edwards | Fall River | June 12, 1945....70 |
| 1923 | Vance, Michael Edward | Pawtucket, Rhode Island | May 5, 1945....75 |
| 1901<br>1920 | Warren, Alva Harding | Malden | November 19, 1945....71 |
| 1906 | Whelan, Charles | North Cohasset | May 29, 1945....68 |
| 1872 | †White, Levi | Worcester | December 1, 1945....96 |
| 1899 | Williams, Hugh | Boston | February 22, 1945....72 |
| 1899 | †Woods, Charles Edwin | Fitchburg | October 12, 1945....82 |
| 1877 | Woodward, Samuel Bayard | Worcester | January 29, 1946....92 |
| 1914<br>1932 | Worthen, Clarence Field | Boston | August 22, 1945....65 |
| 1939 | Wright, Rebekah | Brookline | March 29, 1945....72 |
| 1907 | Young, James Herbert | Newton | February 18, 1946....63 |
| 1924 | Zielinski, Lt. Comdr. Ignatius (MC), U.S.N.R. | "In a plane crash, near Beacon, N. Y." | November 11, 1945....45 |

†Retired fellow.

Total number of deaths of active fellows ..... 58
Total number of deaths of retired fellows ..... 27

Grand total ..... 85

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# CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

## Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

BENJAMIN CASTLEMAN, M.D., *Associate Editor*

EDITH E. PARRIS, *Assistant Editor*

## CASE 32381

### PRESENTATION OF CASE

A sixty-four-year-old widow, a railroad clerk, entered the hospital because of unexplained fever.

The patient had apparently been quite well until six weeks before entry, when she developed a painful, patchy erythematous, nonvesicular eruption on the left shoulder posteriorly that persisted for ten days without other symptoms and then disappeared. Following this episode she noted sharp pains and occasional ringing in the left ear. She was treated with a sulfonamide by a physician, and the symptoms cleared without residua. The patient then resumed her work. Three weeks before entry she began to have localized pain and exquisite tenderness in the nares. The area of painful inflammation progressed rapidly, and in two days the malar and nasal aspects of the face were markedly swollen, painful and red. She also developed fever and called her physician, who treated her with a mixture of oral penicillin and Amphogel for four days. Nevertheless, the lesions progressed to involve the eyes, which became red and "bloodshot" and began to burn. The temperature continued, ranging from 100 to 101°F., and was accompanied by a slight headache and anorexia. Two weeks before entry the face involvement had subsided, but the sides and back of the neck, the temporomandibular joints, the occipital region of the scalp and the ears were affected. These areas were hot and red, and in the suboccipital region a small egg-sized mass developed subcutaneously. The condition grew worse until the patient was partially deafened and was awakened nightly by sharp severe pains, especially in the back of the neck, and occasional twinges in both ears. She had lost about 10 pounds during the six weeks before admission.

As an adolescent the patient had had an attack of erysipelas, during which she became quite sick and developed corneal ulcers. Following this illness aching pains in the shoulders had developed. Ten years before entry the patient had spent nine months at a tuberculosis sanatorium because of pleuritic pain in the left chest and weight loss. The tongue had been sore for about two years. Occasional episodes of urinary frequency, burning and gross hematuria had also occurred during the preceding few years, particularly during the previous six months.

Physical examination revealed a thin woman in no obvious distress, although she complained of tenderness in both temporal regions and along the sternomastoid muscles. The eyes showed old peripheral corneal scars. The ear canals and margins of the drums were somewhat inflamed, particularly on the left. The Weber test was lateralized to the left, and bone conduction was better than air conduction on both sides. The mouth was fissured at the angles, and the tongue was beefy red and sore, with coarse papillae. There was general swelling and slight lymphadenopathy in the region of the left sternomastoid muscle. The lungs were clear, except for a few moist rales at the left base. The heart did not appear enlarged. A blowing systolic murmur was heard at the apex but was not transmitted into the axilla. There was also a soft, blowing systolic murmur over the aortic area. The sounds were regular and of good quality. The abdomen was normal except for slight epigastric tenderness.

The temperature was 101°F., the pulse 100, and the respirations 24. The blood pressure was 160 systolic, 90 diastolic.

Examination of the blood revealed a hemoglobin of 12.5 gm. and a white-cell count of 12,600, with 86 per cent neutrophils, 3 per cent lymphocytes, 5 per cent monocytes, 4 per cent eosinophils and 1 per cent basophils. The sedimentation rate was 6 mm. in 1 hour. Blood Hinton and Widal reactions and a brucellergin test were negative, as were five blood cultures. The urine had a specific gravity of 1.014 and showed a ++ test for albumin; the sediment contained 5 red cells and 25 white cells per high-power field.

An x-ray film of the chest showed only irregular areas of calcification about the right hilus and in the right apical region. Examination of the sinuses, a gastrointestinal series and intravenous pyelogram were all negative. A Graham test showed a poorly functioning gall bladder.

The patient was placed on a high-vitamin, high-calorie diet but failed to improve. On the fifth hospital day an episode of urinary frequency and burning, lasting for several hours, occurred. A urine culture was negative, and no acid-fast organisms were seen in a smear. Sulfathiazole was given. On the seventh hospital day a scratchy sound in late midsystole was picked up in the third and the fourth interspace at the left sternal border. Pressure on the bell of the stethoscope brought this sound closer to the ear. The patient was then taken off sulfathiazole and started on salicylates. For the next several days she appeared subjectively improved, although she complained that the deafness was becoming worse. On the tenth hospital day an electrocardiogram was reported to be within normal limits. An

erysipeloid lesion about the right ear was again prominent, and penicillin therapy was begun. On the twenty-second hospital day she developed an urticarial rash, which disappeared after penicillin was stopped. Five days later bilateral shoulder pain, with swelling and tenderness on the left, occurred and lasted for three days, being followed successively by a hot, swollen, tender right wrist, a painful, swollen left infrapatellar tendon, a tender left wrist, pain over the scapula region of the back, increased epigastric pain and tenderness with spasm and an attack of numbness in the fingers and thumb of the right hand. The patient continued to suffer from severe malaise, listlessness and anorexia. The pericardial friction rub remained constant. The temperature usually rose to 101° or 102°F. in the evening. The white-cell count ranged from 13,000 to 30,000. The sedimentation rate reached 50 mm. in 1 hour. Repeated urine examinations showed + or ++ tests for albumin and 5 to 100 red cells per high-power field. Tenderness in the left costovertebral angle was also noted. The patient grew progressively worse. Several transfusions of whole blood brought about only temporary improvement.

On the fifty-second hospital day a pronounced gallop rhythm was heard over the entire precordium. The neck veins became distended. Cyanosis and puffiness of the face were noticeable. Crackling rales were heard at both lung bases. The liver edge was percussed four fingerbreadths below the costal margin. There was slight pitting edema over the sacrum. Nevertheless, the patient was not dyspneic or orthopneic. The pulse was 110, and the blood pressure 175 systolic, 105 diastolic. Repeated blood chemical studies revealed a nonprotein nitrogen of 107 mg. and a total protein of 5.9 gm. per 100 cc. On the fifty-fourth hospital day, the patient complained of memory loss and pain in the abdomen. The heart was markedly enlarged to percussion, with the point of maximum impulse in the axilla, and a pronounced to-and-fro friction rub was heard over the entire precordium. The urinary output dropped to 60 cc. The patient became stuporous, developed muscular twitching and died on the fifty-eighth hospital day.

## Differential Diagnosis

Dr. Walter Bauer: Did the pain arise in the temporomandibular joints or the adjacent muscles?

Dr. Tracy B. Mallory: I do not know.

Dr. Bauer: Was it erysipelas or an erysipeloid lesion that developed?

Dr. Mallory: The word "erysipeloid" was used.

Dr. Bauer: Was the egg-sized mass a lymph node?

Dr. Wyman Richardson: I saw this patient once but not at that time. I assume that it was a lymph node.

Dr. Bauer: I wonder if anyone who saw the patient would be willing to say whether or not the pain followed the course of the arteries.

Dr. Richardson: I did not observe it.

Dr. Bauer: If I ask any more questions you will know the diagnostic possibilities that I am entertaining.

I interpret the deafness as being of the middle-ear type, but more marked on one side. The Weber test as reported does not rule out bilateral nerve deafness. Were urinary cultures and guinea-pig inoculations for acid-fast organisms done?

Dr. Mallory: There is no record of such tests.

Dr. Bauer: I assume that the beneficial effect of salicylates was short lived.

Dr. Mallory: That is correct.

Dr. Bauer: Does anyone know the duration of the circulatory changes described? Were they neurogenic in origin?

Dr. Richardson: Again, I must say that I saw the patient before that episode and that I do not know.

Dr. Bauer: Will Dr. Lingley show the roentgenograms of the chest and the intravenous pyelograms? I doubt if the gastrointestinal series will be of interest. I assume that the pulmonary findings described were the result of an old acid-fast infection.

Dr. James R. Lingley: The film of the chest as described shows areas consistent with old tuberculosis of the apex and hilus. The heart is normal. There is evidence of old pleurisy at the left base. On the pyelogram there is excretion from both kidneys, and no abnormalities are demonstrable. On this film the Graham test shows a rather faint shadow.

Dr. Bauer: On the basis of this information one wonders if this patient had one or several diseases and when the illness or illnesses began. According to the record, she spent nine months in a tuberculosis sanatorium nine years before admission and was later observed to have pulmonary findings consistent with healed tuberculosis. The genitourinary symptoms, which had been present for at least two years, could have been caused by an acid-fast infection, as the physician in charge suspected. The cutaneous lesions could have been those of erysipelas, and the subsequent serous membrane involvement a manifestation of an ensuing rheumatic fever. Such a sequence of events would account for all the recorded findings. I prefer to reason, however, that the patient suffered from a constitutional disease due to widespread vascular lesions that was first demonstrated by renal involvement. Perhaps I reason this way because of my interest in a special group of diseases. The second manifestation of this patient's illness — the appearance of cutaneous lesions, first over the posterior aspect of one shoulder and later involving the face and neck — is in accord with this concept. Such cutaneous lesions are seen in both disseminated lupus erythematosus and dermatomyositis, but more frequently in the former. Whether the muscles of the face and neck were involved is difficult to

ascertain from the clinical record. At the time of admission the patient had fever, and there was evidence of serous-membrane involvement. Subsequently she developed a migratory arthritis involving a number of articulations and experienced recurring attacks of abdominal pain. This train of events is readily accounted for on the basis of a disease characterized by widespread vascular lesions, such as lupus erythematosus disseminatus, dermatomyositis or periarteritis nodosa. The choice of the proper diagnosis is always difficult, because experience has shown that there may be much overlapping of both clinical and pathological findings in cases of this type. During the last ten years we have seen a number of patients with clinical findings not unlike those observed in the case under discussion. In each case the clinical diagnosis of lupus erythematosus disseminatus was agreed to by Dr. Mallory on the basis of the pathological examination. The exact cause of the cardiac enlargement, which was observed during the last days of the patient's life, is difficult to establish. It could have been a pericardial effusion, diffuse cardiac enlargement or right-sided cardiac enlargement secondary to periarteritis nodosa of the pulmonary vascular system. Such enlargement has been observed in lupus erythematosus disseminatus. I cannot prove its presence in this case, but it is a good possibility.

The laboratory findings are of little help. A white-cell count of 13,000 to 30,000 has been noted in lupus erythematosus disseminatus, particularly in the terminal stages of the disease. Leukopenia is thought by some to be a characteristic finding. In our experience, however, it occurs in only about 50 per cent of the cases of disseminated lupus. I have always been struck by the fact that many cases of periarteritis nodosa have a much higher white-cell count than one would anticipate on the basis of the fever.

In summary, I shall say that this was a case of disseminated lupus erythematosus. I shall not be surprised if Dr. Mallory reports the finding of vascular lesions that were indistinguishable from those of periarteritis nodosa.

DR. RICHARDSON: I saw this patient in consultation once after she had been in the hospital two weeks and before a great many terminal events had occurred. I also do not know what the diagnosis is. At the time I saw her I thought she had a peculiar lack of immunity to a streptococcal infection that could have been erysipelas. After hearing the story my opinion is that she had this infection and that she developed nephritis and pericarditis as a terminal event.

DR. BAUER: Let us not lose sight of the fact that genitourinary symptoms and gross hematuria had been present eight to twenty months before the erysipelas or erysipeloid lesions appeared.

DR. RICHARDSON: Nephritis may have been the first sign.

DR. BAUER: As a general rule one is on much safer ground in assuming that a patient of this age has several diseases. There can be no doubt that this patient had renal disease. Whether it was an independent pathologic process or a manifestation of a systemic disease, Dr. Mallory will soon tell us. I have already committed myself, and I shall stick to my diagnosis of lupus erythematosus disseminatus.

DR. RICHARDSON: I think that you are going to be wrong.

DR. BAUER: It will not be the first time. It is important to realize that the patient's sex and age do not rule out the presence of widespread vascular disease of the type discussed.

DR. LEWIS K. DAHL: Do you think that this patient's age is a little more in favor of periarteritis nodosa than disseminated lupus?

DR. BAUER: No, I do not. On reviewing the cases recorded in the literature one finds that the disease occurs in any age group. Therefore, when one is concerned with a single case, the age of the patient is of no diagnostic help. The same fact is true of sex in both disseminated lupus and periarteritis nodosa. Eighteen per cent of cases of the former occur in men.

### CLINICAL DIAGNOSIS

Periarteritis nodosa?
Lupus erythematosus?

### DR. BAUER'S DIAGNOSIS

Lupus erythematosus disseminatus, with widespread vascular lesions of type seen in periarteritis nodosa.

### ANATOMICAL DIAGNOSES

**Periarteritis nodosa.**
**Multiple infarcts of liver and kidneys.**
Pericarditis, fibrinous, mild.
Mural thrombus of right auricle.

### PATHOLOGICAL DISCUSSION

DR. MALLORY: The autopsy in this case showed a pericarditis less marked anatomically than might have been expected from the clinical evidence. The heart was slightly enlarged, weighing 390 gm. The endocardium and valves were normal, but there was a thrombus in the right auricular appendage and a moderate degree of coronary sclerosis, without occlusion in any vessel. The pleural cavities contained 500 cc. of fluid on each side, but showed no evidence of inflammation. The liver was quite peculiar. Both the external and the cut surfaces were irregularly mottled with focal dark areas 5 to 10 mm. in diameter. In each of these areas the centers of the lobules were large, red and depressed, as in chronic passive congestion. This is the characteristic gross picture of infarction in the liver, which is rarely complete because of the double blood supply and because only those cells in the center of the lobules are killed. The kidneys likewise showed multiple infarcts — in fact, they were so numerous that the picture was suggestive of diffuse cortical necrosis,

although the process was a little too spotty and there were some uninvolved areas. The spleen was grossly normal. On the basis of the multiple areas of infarction in the liver and the kidney it seemed to me that a diagnosis of periarteritis nodosa was inevitable.

When the microscopic sections came through we found vascular lesions in many areas. There was rather marked involvement of the bronchial arteries, although the patient had never had any pulmonary symptoms. Many vessels in the liver and the spleen and the kidney showed lesions of various ages and character. In some the process seemed to be primarily endarteritis, in others occlusive thrombosis, and in a few areas typical periarteritis nodosa, with necrosis of the media, aneurysm formation and so forth. One section from the deltoid muscle showed a typical periarteritic lesion. There was nothing in the case that particularly indicated a diagnosis of lupus, although there was periarterial fibrosis in the spleen, which is consistent with it. The kidney lesions were not suggestive. In several sections from the brain we were unable to find arterial lesions.

DR. BAUER: Did the patient ever exhibit jaundice?

DR. MALLORY: It was never noticed.

DR. RICHARDSON: What do you think of the relation of sulfonamide therapy to this picture?

DR. BAUER: I do not know. So far as one can judge from the record the first erythematous lesions appeared prior to sulfonamide therapy. On the basis of past experience and recent experimental studies I should not administer sulfonamides to a patient suspected of having widespread vascular disease of the type mentioned. Whether sulfonamides were a cause in this case, I cannot say.

DR. RICHARDSON: You do not believe that they were a cause.

DR. BAUER: No, for the reasons previously mentioned.

This case, like others we have seen, illustrates the difficulties the clinician may have in making an accurate anatomical diagnosis. We have seen patients in this hospital who presented the classic textbook picture of disseminated lupus, and yet Dr. Mallory made a diagnosis of periarteritis nodosa on the basis of the microscopic findings. The onset of the disease in this patient resembled both disseminated lupus and dermatomyositis. The subsequent course was more like that of disseminated lupus or periarteritis nodosa. Was this another overlapping case of the type mentioned? I know of no similar case of periarteritis nodosa on record, although Keil* reported the appearance of a red, painful, cutaneous lesion as the first manifestation of the disease in 1 case. Can it be that these diseases, which simulate one another so closely, represent different manifestations of a common pathogenesis? Have you ever seen a case like this, Dr. Ropes?

DR. MARIAN ROPES: No.

DR. MALLORY: There is no evidence to add from the anatomical point of view. There was a wide variety of vascular lesions, some acute, some certainly of months' duration, and some possibly going back two years, although I cannot say that definitely.

*Keil, H. Conception of lupus erythematosus and its morphologic variants, with particular reference to "systemic" lupus erythematosus. *Arch. Dermat. & Syph.* 36:729-757, 1937.

## CASE 32382

### PRESENTATION OF CASE

A seventy-one-year-old retired shoe laster entered the hospital because of bloody urine.

Seven years before entry the patient had had an attack of acute urinary retention, and a suprapubic prostatectomy had been performed at another hospital, following which intermittent painless hematuria and the occasional passage of small blood clots had begun. A year later, cystoscopy was performed at the same hospital and a section of tissue removed from a "growth in the bladder." This procedure effected marked improvement, although the urine continued to be occasionally bloody. Three months before entry almost daily hematuria suddenly began, with frequent passage of clots. Two months later cystoscopy in the Out Patient Department disclosed no source of the bleeding. An intravenous pyelogram showed an enlarged left kidney that failed to excrete the dye during the twenty-minute observation period; the right kidney appeared normal. The bones of the pelvis showed a coarse trabeculation and thickening of the cortex. Shortly thereafter the patient had an episode of urinary retention and was kept on constant drainage at the other hospital for four days. He also developed chills, nausea, vomiting and pain in the left flank and lost 10 pounds in weight. These symptoms persisted. Three days before entry the patient was again cystoscoped in the Out Patient Department, and bright-red blood was seen to come from the left ureter even when the catheter was inserted up to 25 cm. Considerable reddening and some small nubbinlike projections were found about the left ureteral orifice. A retrograde pyelogram did not give satisfactory filling of the left kidney pelvis. The kidney calyxes appeared enlarged, and there was a suggestion of a rather large, smooth, filling defect against the neck of the upper calyx and the pelvis. The kidney outline was not clearly seen on any of the films.

Physical examination on admission revealed an increase in the anteroposterior diameter of the chest, as well as poor expansion. The heart was normal. A large, warm, tender mass, with a somewhat softer center than normal, was present at the left costovertebral angle and extended into the left flank. The entire left side of the abdomen was moderately tense and tender. The liver appeared to be somewhat enlarged.

The temperature, pulse and respirations were normal. The blood pressure was 135 systolic, 80 diastolic.

Examination of the blood revealed a hemoglobin of 75 per cent and a white-cell count of 10,600.

The urine, which was amber colored and acid, had a specific gravity of 1.012 and gave a +++ test for albumin. The sediment contained 250 red cells and 250 white cells, with many clumps, per high-power field. Culture revealed abundant colon bacilli. X-ray examination of the chest was negative.

A biopsy of the bladder was reported as showing malakoplakia.

During the first week in the hospital the mass in the left costovertebral angle became larger, warmer and definitely fluctuant in several places, although the patient had no fever and only a slight leukocytosis. Aspiration produced 4 cc. of brownish pus that showed a slight growth of colon bacilli and aerobic gram-positive bacilli.

On the tenth hospital day an operation was performed.

## Differential Diagnosis

Dr. Fiorindo A. Simeone: This patient had had painless hematuria for seven years, but the presenting symptoms when he was admitted to the hospital were chills, nausea, vomiting, pain in the left flank and weight loss. The physical examination showed the usual signs of inflammation in the left flank. In addition, brownish pus was aspirated from the mass in the flank. A diagnosis of perinephric abscess can therefore be made without much question. Incidentally, the physical examination and x-ray studies suggest that there was no gross lesion other than that in the genitourinary tract. The slight enlargement of the liver was probably of no significance. The heart and lungs were normal on x-ray and physical examination. The peripheral edema could have been the result of malnutrition during the month or more prior to admission, and it could have been associated with hypoproteinemia caused by the loss of blood and protein in the urine during the long period of illness.

Patients with perinephric abscesses can be classified in two main groups — simple and complicated. The distinction is important because both etiology and prognosis are different in the two groups. Simple perinephric abscess is the type secondary to metastasis of organisms from foci of infections elsewhere, such as carbuncles, furuncles and severe tonsillitis. The usual organism is *Staphylococcus aureus* or the streptococcus. The complicated type, which has a relatively poor prognosis, is encountered in patients who, as a rule, have had long-standing infection in the kidney that has spread through the renal parenchyma and into the perinephric space. Later, such infection may actually perforate Gerota's capsule and point in the costovertebral angle. The symptoms and signs recorded in the history of this case are consistent with a diagnosis of complicated perinephric abscess. The patient had gross infection in the left kidney, as manifested by the presence of red cells and large numbers of white cells in the urine and by the culture of colon bacilli — presumably the same organism cultured from the pus aspirated from the flank. No function could be demonstrated in the left kidney by excretory roentgenography.

What was the cause of the pyelonephritis in this case? Seven years before admission a suprapubic prostatectomy had been performed after acute urinary retention that was presumably caused by benign hypertrophy of the prostate. It is possible, on the other hand, that the retention developed because of the presence of blood clot or tissue fragments in the bladder. These could have arisen from a malignant tumor of the kidney or ureter, but there is no information to help settle the question and it should be assumed that the prostatectomy was done for benign hypertrophy. The state of the kidneys at that time is not mentioned. It is customary to investigate the kidneys before prostatectomy, particularly after an episode of acute retention, but the record does not state whether such examination was made; consequently, one is not justified in assuming that the left kidney and ureter were normal.

Dr. Benjamin Castleman: No pyelograms were done at the other hospital.

Dr. Simeone: It is natural to ask if the present illness could have been a late sequela or complication of suprapubic prostatectomy. The pyelonephritis and subsequent perinephric abscess could have resulted from chronic obstruction of the urinary tract. Massive hemorrhage is possible but rather infrequent. The process is bilateral and involves the ureters. The x-ray studies done in this hospital do not suggest such involvement.

The most significant fact is the painless hematuria for seven years in a seventy-one-year-old man. It is reasonable to assume that whatever caused the hematuria was responsible for the pyelonephritis and for the extension of the infection into the perinephric tissues. The record states that blood was seen to come from the left ureter. That excludes from the causes of hematuria the numerous associated systemic conditions and limits the possibilities considerably. In addition blood was flowing not only from the ureter but also from the renal pelvis, because it was obtained from the ureteral catheter that was passed 25 cm. — well into the renal pelvis. The additional information is that cystoscopy revealed no cause for the bleeding in the bladder. If a papilloma of the bladder had been present at cystoscopy and operation six years previously, and if a small section of the lesion had been removed, there should have been residual evidence of the lesion at examination in this hospital. There was reddening around the ureter, with nubbinlike projections about the ureteral orifice, a biopsy of which showed malakoplakia. Perhaps Dr. Castleman will tell us more about the histology of this lesion. Attempts have been made to relate malakoplakia to tuberculosis or mycotic infection in the kidney, but the evidence is not generally accepted and about all that one can say about malakoplakia

is that the condition is associated with chronic and prolonged infections in the kidney, ureter and bladder.[1, 2]

This brings the discussion to the point that is usually reached in trying to determine the cause of hematuria, and help from the roentgenologist is needed. The record states that there was a filling defect in the renal pelvis and neck of the calyx. It might be interesting to have Dr. Schulz show the films.

DR. MILFORD D. SCHULZ: These films, which are representative of the two examinations made, show a definite pressure defect on the calyxes, which are rather ragged in outline and larger than usual. The kidney outline is not visible. There is no calcification in the kidneys, merely dye extravasated into the destroyed kidney parenchyma. The ureter is not visible.

DR. SIMEONE: Is there any irregularity in the calyx itself at that point?

DR. SCHULZ: I do not know. Certainly it looks as if something is compressing it. A lateral film would be helpful.

DR. SIMEONE: Can you be certain whether the filling defect is caused by a lesion outside or within the pelvis?

DR. SCHULZ: I think that it is probably outside the pelvis, but whether or not it is outside the kidney I am not sure. It seems as though the pelvis is being pressed on by some mass.

DR. SIMEONE: Is this calyx dilated?

DR. SCHULZ: All are wider than they should be.

DR. SIMEONE: Is there any evidence of nonopaque stone obstructing the ureteropelvic junction?

DR. SCHULZ: None that I can see. This is Paget's disease down here in the pelvis, that is the only part of which I am sure.

DR. SIMEONE: One can explain the trabeculations in the bones of the pelvis, as Dr. Schulz has said, on the basis of Paget's disease. I do not know any way of connecting the bone disease with the renal lesion. In reviewing some of the possible causes of hematuria in this case, the record of brown pus from the abscess and the slight enlargement of the liver suggested amebic abscess, but I do not know of amebic abscesses that occur in the kidney as they do in the liver. The roentgenograms and the characteristics of the aspirated pus are not consistent with actinomycosis or tuberculosis, which could account for both the hematuria and the chronic infection resulting in the malakoplakia. Tuberculosis as a cause of painless hematuria is unusual in a patient of this age. Painless hematuria occurs early in tuberculosis of the kidney, and renal tuberculosis is generally found under the age of forty. I do not believe that there is evidence of stone, opaque or nonopaque.

The diagnosis of tumor and infection therefore remains. The only question remaining is what type of tumor was present. If the hematuria had been due to this tumor and had lasted for seven years, malignant tumor, although possible, would be unusual. The roentgenogram is consistent with either a malignant tumor or a benign cyst. If the latter had been the lesion involved, it is conceivable that the aspiration of brownish fluid would have come from the cyst rather than from a perinephric abscess. All the findings, however, favor perinephric abscess. My diagnosis is tumor of the kidney, probably a cyst, with perinephric abscess and acute and chronic pyelonephritis.

DR. WYLAND F. LEADBETTER, JR.: We worried for some time about the diagnosis in this case. A fluctuant mass was present in the flank from which we aspirated pus, on the basis of which we believed that the patient had a perinephric abscess. We were unable to ascribe a definite diagnosis to the kidney lesion itself. The main criticism up to this point is that we did not have the necessary x-ray films. We should have had oblique and lateral films, which would probably have given considerable help.

### CLINICAL DIAGNOSES

Perinephric abscess, possibly secondary to tumor of kidney.

Pyelonephritis, acute and chronic.

### DR. SIMEONE'S DIAGNOSES

Perinephric abscess, secondary to tumor of the kidney, probably benign cyst.

Pyelonephritis, acute and chronic.

### ANATOMICAL DIAGNOSES

**Perinephric abscess secondary to renal-cell carcinoma.**

Pyelonephritis, acute and chronic.

### PATHOLOGICAL DISCUSSION

DR. CASTLEMAN: At operation the surgeon found an abscess on the posterior aspect of the kidney over Gerota's capsule. A large quantity of purulent material was drained from it. Further exploration of the kidney, however, showed that its posterior aspect, beyond this abscess, contained a great deal of necrotic material, which grossly suggested a tumor. For that reason the entire mass was removed after the incision was extended upward for a considerable distance. The mass, which was removed in two pieces, because it was so necrotic, proved to be an unusual renal-cell carcinoma. It was quite slowly growing, and it is not inconceivable that this neoplasm had been present for seven years. We have had a number of cases in which the tumor was present for a long time.

The patient was seen yesterday afternoon at the Tumor Clinic, and the note by the physician who saw him states that there had been no further hematuria, the patient felt fine, the urine was clear, and nothing could be felt in the flank.

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## MATERNAL AND CHILD WELFARE

The altercation between Senators Taft and Murray during the recent hearings on the Wagner–Murray–Dingell Bill, although an unfortunate reflection on the dignity of the highest legislative body in the land, had the merit of dramatizing an encouraging phenomenon in the midst of discord: the need for better medical, hospital and nursing care for all people in the United States is recognized by all parties. Disagreements represent conflicting opinions regarding methods and are not concerned with denying that improvement must be provided, the opponents of the bill objecting to any extension of government paternalism and interference and the supporters claiming that only through legislation can better care for all classes of citizens be guaranteed. If the latter ideology is not to prevail, the medical profession must accept the responsibility of inaugurating the reform that is unanimously demanded.

A crucial part of any program, compulsory or voluntary, for the extension of medical care is adequate provision for the health and welfare of mothers and children. Numerous flaws in the Government's attempt to remedy the existing shortcomings in this field — the Pepper Bill — have been pointed out.[1] This is not to say that the objectives of the bill, as stated in the introduction, do not have the full approval of the medical profession. But the methods by which those objectives are to be achieved represent not only a threat to many basic ideals and principles of the profession but also, in many respects, an inefficient and unworkable approach that may well work to the disadvantage of the classes that the bill is designed to benefit.

This and similar legislation offers a challenge to the medical profession. If government interference is to be averted, the inadequacies and inequities of maternal and infant care in many areas, particularly among indigent classes, must be recognized and corrected by the concerted effort of physicians. The following factors in the care of all mothers and infants must be given special consideration: the need for obstetricians qualified by training and experience to provide adequate care in pregnancy, labor and delivery, as well as post partum; adequate nursing care and instruction to prepare the new mother for the duties of parenthood; access to special consultation services in the event of complications; hospitals with sufficient staffs and facilities to furnish continuous maternal and infant care; and the benefit of the newest and best developments in diagnosis and therapy, regardless of cost. A notable example of what can be accomplished by a voluntary approach to the problem is provided by the Chicago Lying-in Hospital.[2] During the past thirteen and a half years 47,945 obstetric patients were cared for at this institution, with 81 deaths, a mortality rate of 0.17 per cent, *but* during that period the mortality rate fell from 0.42 per cent in 1931 to zero in 1944. Undoubtedly such an outstanding achievement cannot be obtained in

all parts of the country, but it can be and is being approached. The pattern of the Blue Cross and the Blue Shield should be extended to include all sections of the country, all classes of the population and all types of medical care: existing programs of prepayment hospital and medical care offer proof that the medical profession can take the lead in co-operation for the public welfare.

Democracy often works in strange and wondrous ways, the existence of inequalities being a major paradox. In many states the Negro is disenfranchised, and efforts to remedy the injustice are met with opposition and terrorization of the subject race; in other parts of the country the necessities of life and health are beyond the reach of a large segment of the population. When such inequalities are discussed in the halls of Congress and, what is more important, constructive attempts at correction are undertaken by the groups most intimately concerned, the fullest benefits of our way of life offer promise of realization. The role of physicians in these efforts is vital.


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## DIAGNOSIS OF MALARIA

Two papers appearing elsewhere in this issue of the *Journal* serve as useful examples to point out problems that confront civilian physicians in diagnosing and treating malaria. These are occurring with sufficient frequency to warrant emphasis on methods for their solution. Three primary situations exist. Discharged veterans or men on inactive duty who were exposed to infection in malarious areas while under suppressive treatment are likely to come down with delayed primary attacks of vivax malaria. A previous history of malarial attacks caused by *Plasmodium vivax* is the basis for subsequent relapses over a period of one to three years. Finally, malaria may develop in a patient receiving transfusions of fresh or stored whole blood from donors who are infected with benign tertian or quartan malaria. No figures are available for the actual frequency of each of these situations. Nevertheless, acquaintance with the underlying facts obtained from the extensive experience of others is pertinent to physicians who are likely to be consulted by persons who served in endemic malarious areas.

The protean character of primary attacks of malaria is well illustrated by London and Most in their warning about differential diagnosis. Bacterial and certain viral infections must be distinguished from malaria. Abdominal symptoms simulating an acute surgical abdomen may also occur during the first few days of clinical malaria. The number, type and intensity of symptoms of primary malarial attacks unfortunately are seldom typical in the textbook sense. Thus, before a diagnosis is made on clinical evidence and treatment is begun, the blood should be thoroughly examined for malarial parasites. The immediate prescription of chemotherapy with sulfonamides or antimalarial drugs without adequate search for parasites only prolongs the time required to make a positive diagnosis and administer curative treatment.

The search for plasmodia before the intervention of chemotherapy frequently fails because of the low incidence of the organisms during the beginning of the primary clinical attack. As few as ten parasites or less per cubic millimeter are sufficient to cause the first febrile attack.[1] According to the figures of London and Most, 54 per cent of the primary attacks and 31 per cent of the relapses with *P. vivax* had less than a thousand parasites per cubic millimeter of blood or approximately one parasite for five thousand erythrocytes. Thus, to request a routine laboratory examination for malarial parasites is not enough. The diagnosis should be made by persons with experience in the improved thick-film and thin-film staining technics and in the specific identification of the parasites. For low concentrations of parasites, the thick-film methods using the Giemsa, Wright–Giemsa or other suitable staining procedures must be employed for satisfactory results: a routine Wright's stain of the blood films does not ordinarily demonstrate the parasites. Failure to find the parasites is usually detrimental to the patient, since it misleads the clinician in prescribing treatment.

If the patient is carrying malarial parasites and

is not properly treated, transmission of the disease to others by anopheline mosquitoes becomes possible. In the northern part of this country, the breeding season for vectors of malaria is limited to the midsummer months. In the midland and southern United States, however, transmission is possible for a greater part of the year. Since Young et al.[2] have proved that native anophelines, *Anopheles quadrimaculatus* and *A. punctipennis*, can transmit malarias of foreign origin and that patients are infectious for anopheline mosquitoes so long as they are subject to relapse, early diagnosis and effective treatment are desirable for control purposes.

That transfusions with whole blood can lead to clinical malaria has been known in this area for many years.[3] In the case cited by Fischer and York transfusion of whole blood stored five to twelve days leads to malaria, and this can be expected to happen more frequently as the methods for preserving whole blood improve. The submicroscopical levels of circulating parasites in these donors with a previous malarial history and the absence of an effective serologic method to detect latent infection leave no alternative other than the rejection of blood from those who have had a past infection, however remote. The relatively long incubation period of transfusion malaria in many cases and the low grade of parasitemia are the features that may confuse the clinician. Here again, blood films should be prepared and examined by skilled technicians to arrive at the correct diagnosis.

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## MASSACHUSETTS MEDICAL SOCIETY

### DEATH

SULZBACH — Wolfgang Max Ferdinand Sulzbach, M.D., of Boston, died recently. He was in his thirty-eighth year.

Dr. Sulzbach received his degree from Rheinische Friedrich-Wilhelms-Universität Medizinische Fakultät, Bonn, Prussia, in 1933. He was a member of the staff of the McLean Hospital.

## MISCELLANY

### PROGRAM ON CARE OF THE HEART

Interest in the control of heart disease is currently being enhanced by the efforts of many professional and lay groups. This attention to heart ailments has long been warranted. The public is becoming more acutely aware of cardiac hygiene than ever before — a growing interest that should be cultivated and guided with judgment as well as vigor.

During the past third of a century, the improvement in mortality from heart disease was most pronounced in the younger age groups and decreased progressively with advance in age. The death rate from diseases of the heart and arteries, corrected for the aging of the population, dropped virtually 30 per cent between 1911–15 and 1940–44, according to experience among the industrial policyholders of the Metropolitan Life Insurance Company. This reduction in mortality from the principal cardiovascular-renal diseases has been particularly marked among white females — 37 per cent in the above-mentioned period. Among males, the decrease in mortality, although not so marked as that among females, was 25 per cent, still a substantial reduction. This still leaves much to be desired in the field of early diagnosis and immediate initiation of adequate cardiac regimes to reduce to a minimum incapacity and mortality from these conditions. Concentration of effort must now be placed on teaching the public what is known about prevention, early recognition and care of cardiac lesions.

To assist in the attainment of this goal, the Metropolitan Life Insurance Company is conducting a special campaign on heart disease during the fall and winter months. At that time, more than 20,000 field representatives, in co-operation with official and voluntary agencies, will reach the homes of millions of policyholders with a recently published pamphlet, "Your Heart," developed in co-operation with the American Heart Association. A lay educational film on heart disease is also being prepared. Distribution will be made to physicians of a packet in which will be included material of special interest to doctors. A scientific exhibit on heart disease, first shown at the meeting of the American Medical Association in San Francisco, is available for state and local professional meetings.

## BOOK REVIEWS

*Periodontal Diseases and Soft Tissue Lesions of the Oral Cavity: Diagnosis and treatment.* By Arthur H. Merritt, M.S., D.D.S., Sc.D. Third edition. 8°, cloth, 256 pp., with 53 illustrations. New York: Macmillan Company, 1945. $3.50.

The third edition of this book has an added section, consisting of six short chapters and nine illustrations, entitled "Soft Tissue Lesions of the Oral Cavity." In the opinion of the reviewer, this section, which gives a superficial and inadequate discussion of lesions of the mucous membrane, with a few rather poor illustrations, detracts from rather than enhances the value of the book, which offers useful practical information — based on extensive clinical experience in the diagnosis and treatment of periodontal diseases — regarding the management in office practice.

Recent advances in the knowledge of periodontal diseases are not referred to, and the bibliography is incomplete and fails to satisfy the inquiring mind of the student or practitioner of dentistry.

*The Psychology of Women: A psychoanalytic interpretation.* Volume II: *Motherhood.* By Helene Deutsch, M.D. 8°, cloth, 498 pp. New York: Grune and Stratton, 1945. $4.50.

Rarely does a reviewer have the pleasure of studying a book of this type. Dr. Deutsch has presented her second volume of this broad and controversial field with a clarity, construction and determined purposefulness that is often lacking in psychologic and psychoanalytic literature. The reviewer highly recommends this book to all professional members of allied psychiatric services.

The text develops the theme of most of the psychologic, really psychoanalytic, factors in maturing and older women. The chapters that seemed to have outstanding informative clarity were those on pregnancy, confinement and lactation and the mother-child relation.

Some features with which the reviewer personally takes issue are offered. In the first place, there seems to be a tendency to generalize on the subject of social stresses, which, of course, makes the text more popularly interesting; this might also be said concerning the subject of matriarchy versus patriarchy. Secondly, as in other psychoanalytic texts, the ascribing of deeper, unconscious motives for scientific investigations in social and anthropologic fields was noted. Considerable discussion could center about the difference between maternal instinct and motherliness (mother love). Dr. Deutsch places emphasis on the latter almost as though it were a separate entity. To the reviewer, motherliness seems to be a more highly developed expression of maternal instinct, with volitions and inhibitions proportionately more highly developed. In this respect the psychologic expression "rich determinants of emotional motherhood" seems to be part of the entire process of adaptation to inner and outer tensions rather than a subject set apart for strictly psychoanalytic study. Another item of issue is that Dr. Deutsch, throughout the text, apparently found difficulty in properly evaluating biologic and physiologic elements and sometimes verbally set them aside, only to bring them up repeatedly in connection with processes unexplainable by psychoanalytic methods. Yet considerable space was devoted to the physiology of the sexual act and organic components thereof. Still another point to be clarified is the thesis that pregnancy is often fortuitous and accidental, but that cultural actions, customs and taboos make for compensatory conscious acceptance of the fetus whereas unconscious factors may be varied and changeable; one might also question the emphasis placed on drives toward achieving motherhood.

A fuller discussion of the psychologic factors in pregnancy and motherliness in Fascist cultures, as well as more consideration of the same subject in Egyptian and Greco-Roman civilizations, would have been of greater value than the devoting of so much space to more primitive social levels and literary subjects.

Considerable positive acclaim is given to the "motherly woman," with the implication that this type of woman is the best for adjustment and mature gratifications. The reviewer again states that the book is professionally adequate, quite informative and effortless to read.

## BOOKS RECEIVED

**The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.**

*Conscience and Society: A study of the psychological prerequisites of law and order.* By Ranyard West, M.D. (Lond.), D.Ph. (Oxon.). 8°, cloth, 261 pp. New York: Emerson Books, Incorporated, 1945. $3.00.

Dr. West believes that world-wide law (government and society) must be based on human nature as it is, not as prejudice makes it out to be, and that laws must be psychologically sound. The work is divided into three parts. The first is a study of human nature, including a discussion of three of its philosophies (those of Thomas Hobbes, John Locke and Jean Jacques Rousseau), human government, as viewed through the ages, the psychologic theories of human nature and the actual behavior of man. The second is a study of the control of human nature by law, comprising chapters on a psychologic theory of law and the stage of social organization represented by present international law. The third part attempts to apply present knowledge of man's social nature to present problems of social organization and world order under the headings of the emotion of loyalty and manipulation of world forces to attain world law.

Dr. West is a psychoanalyst, and under the headings of psychologic theories of human nature and the actual behavior of man he devotes more than a third of his text to psychoanalysis, in which he discusses the principles of psychoanalysis, the findings of Freud in neurosis, Freud's theory of human nature (with case material) and the psychologic theories of human nature that contrast with that of Freud. The behavior of man is discussed under the headings of instinct in action, observations of child psychology, and psychoanalytic reactions and real life side by side. This last chapter is extensively documented with case histories. The book should be useful to all persons interested in psychoanalysis.

*Report of the National Health Survey.* Conducted by Canadian Medical Procurement and Assignment Board. 8°, paper, 336 pp. Ottawa: Edmond Cloutier, 1945.

This extensive detailed report is divided into eight parts: civilia medical manpower, medical schools, public health, hospital personnel and facilities, industrial medicine, war medical services, nursing services and dental services. Various governmental orders and statistical tables are included in a large appendix.

*The Physiology of the Newborn Infant.* By Clement A. Smith, M.D., professor of pediatrics, Wayne University College of Medicine, and medical director, The Children's Hospital of Michigan. 8°, cloth, 312 pp. Springfield, Illinois: Charles C Thomas, 1945. $5.50.

In this monograph Dr. Smith considers the fetal and neonatal physiology of the child in its various aspects. He divides his subject as follows: respiration, circulatory system, blood (with a chapter on icterus neonatorum), metabolism and heat regulation, the digestive tract, nutrition, kidneys, endocrinology and immunology. The text is well documented, a lengthy list of pertinent references being appended to each chapter. The book is well printed on good paper, with a good type. This monograph should be in all medical reference collections and in the libraries of pediatricians and obstetricians.

*Recent Advances in Obstetrics and Gynaecology.* By Aleck W. Bourne, M.A., M.B., B.Ch. (Camb.), F.R.C.S. (Eng.), F.R.C.O.G., obstetric surgeon, St. Mary's Hospital, consulting obstetric surgeon, Queen Charlotte's Hospital, and consulting surgeon, Samaritan Hospital for Women; and Leslie H. Williams, M.D., M.S. (Lond.), F.R.C.S. (Eng.), F.R.C.O.G., obstetric surgeon to out-patients, St. Mary's Hospital, consulting obstetric surgeon, Queen Charlotte's Hospital, the Jewish Maternity Hospital and the Nelson Hospital, surgeon to in-patients, Samaritan Hospital for Women, and examiner in midwifery and gynecology, University of Cambridge and Conjoint Board. Sixth edition. 12°, cloth, 357 pp., with 77 illustrations. Philadelphia: The Blakiston Company, 1945. $5.50.

The first edition of this well known review of obstetrics and gynecology was published in 1926. Successive editions have been issued every few years to the present time. The authors were surprised to find that much valuable research had been done during the last four war years. A few entirely new chapters have been added to the present edition, namely, nutrition in pregnancy, vitamin K, stillbirth and neonatal death and erythroblastosis. There is also a new chapter on x-ray therapy in gynecology, which has been separated from the chapter on radiologic diagnosis. Certain chapters appearing in previous editions have been discarded, such as antenatal care, pyelitis, puerperal sepsis and electrophysical therapeusis. It should be noted that only three chapters remain from the first edition of 1926, all of which have been thoroughly revised and brought up to date. In the light of this fact, all editions of this book should be in every reference collection on obstetrics and gynecology.

## NOTICES

### ANNOUNCEMENTS

Dr. Edward C. Dyer announces the opening of an office for the practice of pediatrics at 330 Dartmouth Street, Boston.

Dr. Benjamin I. Nadelman announces his return from military service and the reopening of his office for the general practice of medicine at 140 Blue Hill Avenue, Roxbury.

Dr. John A. Reidy announces the removal of his office to 1101 Beacon Street, Brookline, for the practice of orthopedic surgery.

Dr. Irving Silverman announces his return from military service and resumption of the practice of pediatrics at 416 Marlborough Street, Boston.

(*Notices continued on page xix*)